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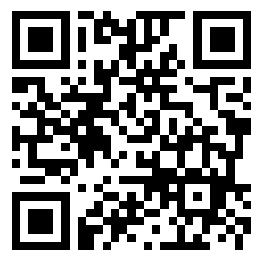

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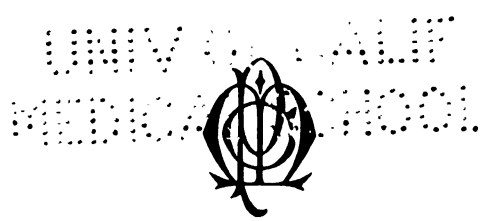

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A CLINICAL LECTURE ON ANEURYSM.

Delivered at St. Bartholomew's Hospital, February 14th, 1902.

By SIR WILLIAM CHURCH, M.D., F.R.C.P., Physician to the Hospital.

GENTLEMEN,—There happen to have been lately in my ward several cases of aortic aneurysm. I shall not go at length into those cases, as I do not think they present any particular features worthy of remark, but I shall rather use them as a text upon which I will make some general remarks upon aneurysm, and more especially upon thoracic aneurysm. With regard to the cases which you have seen in my wards, three were in Mark Ward, and two were cases of ordinary aneurysm of the arch of the aorta, and one a case of abdominal aneurysm. One case of thoracic aneurysm presented, before death, some unusual features in the very large size that it attained. It was an aneurysm of the arch of the aorta, and it projected externally, displacing the clavicle from the sternum, and also rising unusually high in the neck. I will pass round a photograph which was taken of the man shortly before his death. You will see from the photograph that it attained a gigantic size, yet for a long time, as is usual with aneurysms which occur high in the thorax, it did not cause any interference with deglutition; there was no marked pressure upon the œsophagus until a comparatively short time before death. Owing to its huge size it did then begin to press upon the œsophagus, and before death, for ten days or so, the poor man had been unable to swallow. After death we found that the wall of the œsophagus was undergoing necrosis from pressure. But with regard to pressure symptoms in thoracic aneurysm I shall have to say something more hereafter.

Turning to the general remarks which I should like to make upon aneurysm, students used to be,

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and I believe are to some extent now, perplexed by the different so-called varieties of aneurysm, such as the fusiform, the sacculated, and true and false aneurysm. I am happy to say that those terms have to a great extent fallen into disuse. What was meant originally by true aneurysm was aneurysm in which all the coats of the artery could still be traced. False aneurysm was one in which the coats of the vessel, either all of them or the inner and middle coats, had apparently been either destroyed or were not represented over the dilated portion. The terms fusiform and saccular only arose from the shapes. I have had brought down for your inspection a few specimens from the museum to illustrate this. Here is a very good example of what would be called a true sacculated aneurysm of the aorta, which would be still better seen in a smaller vessel. Here you see it in a popliteal artery. Here is an example of fusiform aneurysm, which also, I think, is from the popliteal space. By far the best definition of aneurysm, certainly for practical purposes in large vessels such as the aorta, is that which was given by the late Dr. Hilton Fagge. He describes aneurysm as a circumscribed swelling or tumour containing fluid or solid blood, communicating directly with the canal of a blood-vessel (artery), and limited by the tissues which form the so-called sac. That is the condition of an aneurysm in the majority of instances of large aneurysm. The walls are made up not only by the remains of the outer coat of the artery but also of a hardened sac of condensed tissues, partly formed by pressure out of the existing tissues, and partly the result of changes which have taken place as the aneurysm has advanced. I would allude to the fact that there are aneurysms, however, in other parts of the body to which the terms true and false may be more strictly applied. I mean miliary aneurysms of vessels of the brain. There you have a direct hernial protrusion through the outer coat of the artery of the inner and middle coat of the artery. I feel confident that that is a common condition in miliary aneurysms of the vessels of the brain, and in the same way in aneurysms of the pulmonary artery. In them there generally is continuity of the inner coats of the artery and it is the outer coat which has given way; indeed, that is what you would expect when you consider that the way pulmonary aneurysm is formed is by ulceration of the lung, and therefore the outer wall of the vessel is attacked by the ulceration and destroyed, and then the inner walls make a hernial protrusion, which develops into what we speak of as a pulmonary aneurysm. But with those exceptions in almost all aneurysms the condition of the walls of their sacs is a mixed one, and hence a good many years ago you had not only true and false aneurysms, but also, added to them, a number of what are called mixed aneurysms. I only make these remarks in passing because sometimes those terms have led to difficulty and confusion in students' minds.

There is one other form of aneurysm which I might briefly allude to, and that is not nearly so common as the sacculated one. That is what is known as a dissecting aneurysm, where there is rupture of the inner coats of the vessel but not of the outer, and where the blood escaping from the artery strips off the inner coats of the vessel from the outer coat. Here is a very good specimen of that form, in which you see the coagulated blood between the inner and the outer coats.

I have briefly reminded you of the different forms of aneurysm which we meet with, but I have not exhausted them all, because I have said nothing of the formation of what we now speak of as secondary aneurysms in connection with septicæmia and embolism.

I would now like to make a few remarks upon the ætiology of the disease, that is to say, the conditions under which we find aneurysm occurs. First of all age should be mentioned. Aneurysms in young people, except in connection with emboli from a pyæmic cause, are extremely rare. The period for the occurrence of aneurysms is that of the greatest bodily vigour, and more especially of that period which coincides with the commencement of degeneration. Hence I think we may confidently say that the formation of aneurysm generally comes about from changes within the arterial wall plus strain thrown upon it by the blood, caused by exertion, and especially certain forms of exertion; that is to say, in order for aneurysm to be produced you have not only to have degenerative changes in the wall of the artery but you must have the wall submitted to some unusual strain. I think that is pretty clear, because the degeneration of the arterial walls is stated in middle life to be quite as common in women as it is in men, but yet the occurrence of aneurysm in

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women is a rare event. In fact, the rarity in women of this condition is one of the reasons why one is very doubtful as to the diagnosis of another case which I intend to bring before you. It is the case of a woman in Faith Ward. For some time I was in hopes she had not got aneurysm ; and even now the diagnosis of it is somewhat uncertain. I should be sorry to say absolutely that she has, although, having had the opportunity of watching her for many weeks, I think the probability is that she is suffering from aneurysm, and not, as I trusted, from a tumour of another sort. Aneurysm is very much commoner, then, in males than it is in females in advanced and middle life, that is to say, after forty-five or fifty, although degenerative changes in the walls of the vessels are as frequent in women as they are in men; but, speaking generally, men are subjected to much greater strain in their arterial system than are women, so I think that is strong confirmatory evidence that in the formation of aneurysm not only degenerative changes but superadded strain is required.

Next with regard to the degenerative changes which occur in arteries, which you must look upon as being the first factor in the formation of aneurysms. The first change I should mention would be senile degeneration. A man's arterial system may become older than other parts of his body in the same way as we see that baldness may occur without other parts of the body being affected, or grey hair may appear without signs of senility, and both of them certainly are senile changes. In the same way the portion of the body to be first attacked by senile changes may be the arterial system. This has long been recognised, and there is an old and hackneyed saying that a man is as old as his arteries, a truism which has been well known for many years. In connection with arterial degeneration, whether you call it by the name of endarteritis or arterial sclerosis or by any other term—because I do not really know any difference between them—the changes are undoubtedly associated with certain conditions of body,—what are spoken of generally under the term diathesis, which is a way of recognising a certain bodily conformity or condition, the causes of which which we are yet unacquainted with. We speak of a gouty, a rheumatic, or an arthritic diathesis ; what we know is that gouty people, and rheumatic people and arthritic people are apt to have degenerative changes in their arteries at an earlier period than those who have not presented those conditions which we speak of under the terms of gouty, rheumatic, or arthritic diatheses. But there is one other condition which, there can be no doubt, predisposes very greatly to arterial change, arterial sclerosis, or endarteritis, and that is syphilis. You find in a very large number of cases of aneurysm, both in men and in women, that they have at some previous time suffered from syphilis. Both the soldier, whose photograph has been passed round, and also the sailor who is in Mark Ward have very distinct histories of syphilis. In the case of the woman who is in Faith Ward, there is no very distinct history of syphilis, yet the fact of her having been twice pregnant, and that her first child died of convulsions at two months old, and was a wasting infant, and the other was a dead child of seven months' gestation, although it does not prove that the children were syphilitic, still makes it not improbable. It made it so probable that for a long time I hoped that the evident tumour which existed in her chest might be of a gummatous nature, and I am not prepared altogether to give up that hope still, although it is more probable that it is an aneurysm. The connection of syphilis with the tendency to the occurrence of aneurysm is undoubted, and it is a most important factor.

The recognition of the symptoms of thoracic aneurysm is either extremely easy or, as we see in the case of the woman I have mentioned, exceedingly difficult. When an aneurysm has attained any large size, and has begun to come forward from the chest wall, so that you get visible or palpable pulsation, then there is not generally very much difficulty in making up your mind whether you are dealing with a pulsatile swelling of carcinomatous or other nature or an aneurysm although there is difficulty even then. But in the remarks I make I would rather draw your attention to the symptoms which accompany aneurysm when no pulsation is visible or palpable, and when you have to judge of the probabilities of the nature of the disease that the patient is suffering from from other symptoms. The most prominent is pain. It is rare for aneurysm, at all events aneurysm of any size, to exist for any considerable time without causing very considerable pain, pain which it may

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be difficult to discriminate from cardiac pain or the pain which you get in association with cardiac disease. I think, as a rule, in aneurysms of the ascending or transverse portions of the arch of the aorta you find that the pain is referred to the first, second, or third segments of the thorax rather than to parts lower down, whereas in cardiac pain, and especially in angina pectoris, the pain is often referred to the lower part of the thorax, on a level with the false ribs; this is an important item for you to remember, and it may help you very considerably. Aneurysm may and does exist without there being any cardiac symptoms, that is to say, palpitation is absent, there are no distinct cardiac murmurs, and, of course, there may be no abnormal arterial sounds to be heard. It is quite common for aneurysm to be present without your being able to recognise any abnormality in the sounds in the thorax, although, of course, thoracic aneurysm, especially of the ascending aorta, is frequently accompanied by disease of the aortic valves. The respiratory symptoms are extremely variable. They are generally more marked than are the cardiac. Those aneurysms, which you have the greatest difficulty in recognising, are those generally which are deep-seated and which occur near the spine. Not infrequently you meet with symptoms of pressure upon the trachea or on one of the bronchi without there being any other very obvious sign. Whenever you do meet with those symptoms it is always important to bear in mind the possibility that you are dealing with a case of aneurysm. Pressure on the trachea or bronchi is almost invariably accompanied by a brassy sort of cough, a harsh resonant cough. Whenever you meet with this cough, not only the possibility but the probability of your having to do with aneurysm should be present to your mind. Although it generally occurs later, when the aneurysm has attained a considerable size, you have in connection with the respiratory symptoms often paralysis of one or other of the vocal cords, due to pressure upon the recurrent laryngeal nerve, which, for anatomical reasons, occurs very much more frequently in connection with the left vocal cord than in connection with the right. There you get another symptom which is sometimes present when very few physical signs of aneurysm can be detected, and that is interference with the action of the pupil, especially aneurysms which exert pressure towards the spine. In such cases there is paralysis of the ciliary muscle, what is called a contracted pupil. Dyspnœa, of course, depends a good deal upon the amount of interference with the innervation of the larynx and also upon the amount of pressure upon the bronchi or the trachea. As the disease goes on, and the aneurysm gets larger, you are apt to get secondary changes in the lungs, which I will not go into now.

Dysphagia, which I mentioned in connection with that unfortunate man whom many of you saw in the dead-house this week, is not common where you have the aneurysm projecting externally. It is especially in aneurysms which occupy the posterior part of the chest rather than those which come forward in which you meet with dysphagia, and especially those which are in connection with the transverse arch. Dysphagia is generally due to the direct pressure of the tumour obstructing the œsophagus, but cases have been reported and observed in which it appears that there was difficulty in swallowing owing to pressure upon the vagus where there was no real pressure upon the œsophagus, but in almost all the cases where dysphagia is present it is due to direct pressure of the aneurysm upon the œsophagus.

I do not intend to say anything about the treatment of aneurysm, or, at all events, very little, for, apart from what I may mention as the surgical rather than the medical treatment of aneurysm—I mean the introduction, as you saw in the abdominal case, of wire, or the attempt to do good by galvano-puncture or by scratching the inside of the aneurysm with needles—medically we have but few means of attempting to do good; but the treatment which does do good sometimes is perfect rest, limited diet, and very large doses of iodide of potassium. Since I have dwelt upon the connection of syphilis with aneurysm, you might rush to the conclusion that iodide of potassium does good because it does good in syphilitic disease. I think that is not the reason. The main good that iodide of potassium does, in large doses, especially when the patient is at rest, is that it tends to lower the blood-pressure. Whether it has any effect upon the blood in rendering it more coagulable I will not express an opinion, but just as in heart disease you can get great relief by giving large doses of iodide or iodide and bromide of potassium mixed, so I believe the great value of iodide of

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potassium in the treatment of aneurysm arises from its causing a diminution in the blood-pressure rather than from exerting any specific action upon any syphilitic taint that may be in the body. When the aneurysm has been formed the damage has been done. It is possible that you may, by the iodide of potassium, destroy the toxin, if there be one, which causes this degeneration of vessels in syphilitic cases, so you may prevent the arterial disease from becoming more general. Iodide of potassium certainly does, in some cases, relieve your patients, even when a large sacculated aneurysm has been formed.

I am afraid these have been rather disjointed remarks, because I have chosen far too great a subject for these short clinical remarks, but those who have been in my wards have seen these four cases, three of which were very much alike. The other case has gone to the surgical ward under Mr. Harrison Cripps; at present there seems to be no consolidation in his abdominal aorta; it still apparently pulsates in the same way as before the wire was introduced, but we hope he will eventually derive benefit from the procedure. The case of the woman, which to me is most interesting, is still one in which the diagnosis is not absolutely certain.

Ætiology and Treatment of Pruritus Vulvæ.—Seeligmann ('Deutsche med. Woch.') has been able to isolate and cultivate a diplococcus from all his cases of pruritus vulvæ during the last ten years. It resembles the gonococcus, but takes the Gram stain, and differs also in its method of growth. He found that 10 per cent. guaiacol vasogen killed the cultures of the diplococcus in five minutes, which confirmed the almost invariable success of this salve in his clinical experience. He has the vasogen applied on cotton at night, repeated for several evenings if necessary, and has thus succeeded in curing many cases of secondary and primary true pruritus. If the 10 per cent. strength of the guaiacol vasogen is not sufficient, he uses a 15 or 20 per cent., but avoids this when possible, as it is liable to irritate. If the pruritus returns later, one or two applications of the vasogen will usually dispel it.—*Journ. Amer. Med. Assoc.*, March 22nd, 1902.

HYPEROPIA.

A lecture delivered at the Medical Graduates' College and Polyclinic.

By ERNEST CLARKE, F.R.C.S.,
Surgeon, Central London Ophthalmic Hospital.

Hyperopia (or Hypermetropia).

The hyperopic eye is the flat or undeveloped eye, in which, with accommodation at rest, parallel rays come to a focus beyond the retina (Fig. 1), and only convergent rays focus on the retina, but as in Nature all rays are either parallel or divergent, it follows that the hyperopic eye at *rest* sees everything indistinctly.

The punctum remotum of a hyperope is beyond infinity; it is the point toward which the luminous rays ought to converge in order to be focussed on the retina; hence it is *behind* the eye, and is therefore represented as negative, being the virtual and not the actual focus of the distant rays.

Rays coming from a point on the retina, of course, diverge, and on passing through the dioptric system of the eye emerge in the normal eye as parallel rays; but in hyperopia, although they are not so divergent as they were before refraction, they still diverge if the eye is at rest, and therefore never come to a focus in front of the eye, but if they are prolonged back they will meet at a point behind the eye—R., the punctum remotum (Fig. 1). The more convergent the rays outside the eye are, the nearer will their "backward prolongation" focus, hence the nearer R. is to the eye the higher will be the hyperopia. This is the same as we shall find in myopia, viz. the higher the M. the nearer is R. to the eye, but the difference is that in M., R. is in front of the eye, and in H. it is an imaginary point behind the eye. Thus the degree of H. is in inverse ratio to the distance of the punctum remotum. In myopia we can measure this directly, but in hyperopia we can only do so indirectly by employing convex glasses.

Suppose the p. r. of a hyperope is 33 cm. behind the retina. Now, we are aware that a lens whose focal point is 33 cm. is 3 D., that is, such a lens has the power of converging parallel rays to a point 33 cm. on the other side of the lens, and conversely, rays diverging from a point 33 cm. in front of such a lens become parallel on passing through. If this lens is put in front of the eye

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of this hyperope it will so act that, assisted by the dioptric system of the eye, it will cause parallel rays to focus *on* the retina, hence the measurement of hyperopia is that convex lens which enables the hyperopic eye at rest to see distinctly objects at infinite distance, and the focal distance of such a lens represents the distance of the far point from the eye. In the above example we find that + 3 D. is this glass, and we accordingly say that this eye has a hyperopia of 3 D. Now a hyperope differs from a myope in that he can correct his defect up to a certain point; he can, by accommodation, produce the same effect on parallel rays as if a convex glass were placed in front of the eye. This apparent advantage brings with it many disadvantages, viz. all the trouble incident to eye-strain.

The hyperopic eye is never at rest, it has to accommodate for distance as well as for near objects. The emmetrope's ciliary muscle is at rest when he is looking at anything beyond twenty feet, but the hyperope's eye is never at rest if he attempts to see distinctly; and, moreover, when he wishes to look at a near object he starts with a deficit, which deficit is the amount of accommodation he required for distant vision. Thus a hyperope of four dioptres with five dioptres of accommodation can focus distant objects clearly, but then he has only *one* dioptre left for near vision; this will only bring his near point to one mètre from the eyes. Again, take a hyperope of two dioptres with five dioptres of accommodation; he only has three dioptres available for accommodation of near objects; this brings his near point to 33 cm., but he is using the whole of his accommodative power for this, and it is impossible for him to do this for long without fatigue, and so we get all the symptoms of accommodative asthenopia.

In hyperopia $a = p + r$ [*i. e.* $a = p - (-r)$], therefore $p = a - r$; in other words, the *available* amount of accommodation power is represented by the total accommodation power less the amount required to correct the hyperopia, so that although age for age the hyperopic eye has the same total amount of accommodation power as the normal eye, it has less to use for near vision if uncorrected. In Fig. 3 the amount of available accommodation power in the uncorrected hyperope of 4 D. is represented by the number of divisions between p. and the zero line; thus, at the age of thirty, we find only three divisions representing 3 D. of accommodation power, because, although he has 7 D. total power like the emmetrope, 4 of this is used up to correct his defect; at the age of forty we see that p. crosses the zero line, in other words, all available power is lost; he has 4 D. left, but this is used up in correcting his defect. Beyond this age he loses still more accommodation power, and this means that he cannot even correct his defect, in other words, he cannot obtain clear images of anything far or near. This condition of affairs will obtain, of course, at an earlier age if the hyperopia is higher. Thus a hyperope of 10 D. at twenty-five has only 8 D. accommodation power, and consequently sees everything indistinctly. Such persons very often approach their eyes very near to their work in order to obtain larger retinal images, and an erroneous diagnosis of myopia is liable to be made.

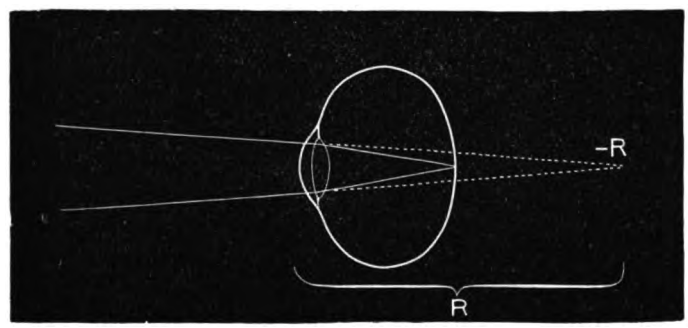

FIG. 1.—Showing the punctum remotum of a hyperopic eye.

The hyperopia which is at once recognised, the patient at once confessing to improved vision with a convex glass, is called manifest, and this manifest

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hyperopia (Hm.) is expressed in amount by the *strongest* convex glass the patient accepts; for instance, a patient sees $\frac{6}{9}$ but with + 1 D. up in front of the eye $\frac{6}{6}$, and + 1·5 D. makes the letters hazy; then + 1 D. = Hm.

Again, when the defect is hidden by the patient using his accommodation and obtaining perfect distant vision, the manifest H. is represented by the strongest convex glass with which he can see as well as with the naked eye. (The emmetrope and myope will not accept any convex glass for distance.)

The latent hyperopia is the additional hyperopia, which shows itself when the accommodation has been relaxed with atropine. If the patient quoted above, when under atropine, sees $\frac{6}{6}$ only when + 3 D. is put up, in his case + 2 D. represents the latent hyperopia (Hl.), the total hyperopia (Ht.) being the sum of Hm. and Hl. Hm. + Hl. = Ht. Thus:

- Total Hyperopia (*Ht.*)
  - Latent H. (*Hl.*) — Only revealed under a mydriatic.
  - Manifest H. (*Hm.*)
    - *Absolute* (*Hma.*), which no amount of accommodation can correct, represented by the weakest convex glass.
    - *Facultative* (*Hmf.*), when distant objects can be clearly seen with or without convex glasses, represented by the difference between the strongest and weakest convex glass.

Besides these varieties we have RELATIVE hyperopia, when parallel rays can be brought to a focus on the retina by accommodation and convergence, the latter being in excess of the amount of convergence required. To accommodate for a near point the patient must converge to a point still nearer, *i.e.* to see clearly at 22 cm. he must converge, say for 12 cm., in other words, he squints.

This want of harmony between the accommodation and convergence efforts is a constant cause of asthenopia. In the normal eye, the two eyes converging for a point one mètre off form a mètre angle, and this is the unit. Eyes converging for a point 50 cm. off have to converge two mètre angles, and so on. Now a hyperope of 2 D., when looking at a point 33 cm. off, is using $\frac{100}{33}$, *i.e.* 3 D. added to the correction of his H., viz. 3 + 2 = 5 D., but he is only converging three mètre angles instead of five, consequently he is using 2 D. of accommodation in excess of convergence.

Now Nature has endowed many hyperopes with the power of increasing their accommodation *to a certain extent* without varying their convergence; this faculty is the result of "nerve education." We shall see when dealing with myopia that the same thing occurs, only in this case it is the convergence that is used in excess of the accommodation. There is a minimum amount of effort when convergence and accommodation work harmoniously together, as it were, supporting each other, but when one is used in excess of the other it has to work unaided and alone, and strain is liable to ensue.

Many hyperopes never learn this trick, as we may call it, of using their accommodation in excess of convergence, and therefore they are less likely to suffer from strain (although the unconscious efforts to do so may induce it); but a worse evil befalls them, they lose binocular vision, and squint. A hyperope under these circumstances finds him-

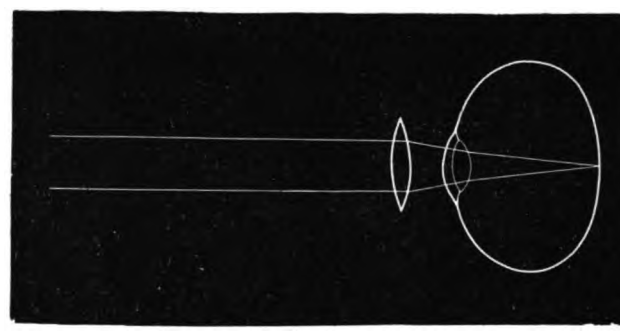

FIG. 2.—Showing parallel rays focussed on the retina of a hyperopic eye by means of a convex lens.

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self in the following dilemma: if he wants to see binocularly he must use less accommodative power than he requires to see distinctly; or if he wishes to see distinctly he must sacrifice binocular vision, which ends in squint. He must choose between distinct vision and binocular vision. Distinct vision is more craved for and more useful than binocular vision, especially if the latter is not quite perfect owing to one eye being more defective than the other; consequently he sacrifices binocular vision and squints. The dilemma ends and asthenopia ceases.

CONDITIONS CAUSING HYPEROPIA.—1. *Axial hyperopia.*—This is the commonest form, and is the condition of most eyes at birth; it is due to the

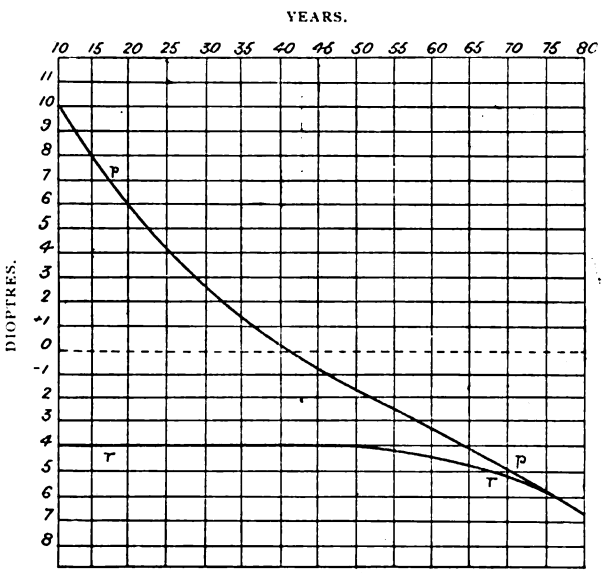

FIG. 3.—Showing the amount of available accommodation power of a hyperope of 4 D. at different ages, represented by the abscissæ between the zero line and "p."

shortening of the antero-posterior diameter of the eye, and may be due to a flattening of the globe or to a general diminution in size. This condition is due to an arrest of growth of the eye, and is often associated with arrest of growth of the bony parts surrounding—thus the face of a hyperope often shows want of relief.

The tendency is for eyes that are hyperopic at birth to grow towards the normal, and even become myopic; but after fifty, owing to the increase in size and the flattening of the lens with age, there is a tendency for all eyes to become hyperopic as life advance—this is called "acquired hyperopia." In Fig. 3 the acquired hyperopia is shown by the downward curve of "r" commencing soon after the age of 50.

2. *Curvature hyperopia.*—Due to a lack of convexity of the refractive surfaces, and is generally associated with astigmatism.

3. *Index hyperopia.*—Due to diminution in the index refraction of the media.

4. Hyperopia may be due to absence of the lens (aphakia), or its total or partial dislocation.

5. Tumours or exudations causing advance of the retina in the eye will cause hyperopia.

SYMPTOMS.—It is the facultative hyperopia which the patient can correct, and thus more or less conceal at will, that is one of the most common causes of eye-strain, and the reason is very apparent.

In absolute Hm. vision is never acute, and the patient makes no attempt to strain his acommodation because he finds the result useless, more or less.

In relative Hm. we have seen that only monocular vision can be acute, and that asthenopia generally ceases when the squint appears; but in facultative Hm. patients are most frequently quite unaware that they are suffering from any defect of the eyes, they can see well at a distance and their near vision is as good as they want, and they have no idea that the *headaches* that come on after near work are caused by eye-strain, and they will, in all probability, be treated for all manner of diseases before the real cause is discovered. It is true that if this eye-strain goes on for long signs of inflammation will often show themselves in the eye and its appendages, such as conjunctivitis and blepharitis, and lead the patient to the eye surgeon, but even he may miss the true cause unless he makes it a rule (which he ought to) to examine the eye under atropine of all young people (and this is essentially a disease of the young) suffering from chronic inflammation of the lids or conjunctiva.

The facultative hyperopia of the young becomes in middle age relative, and after fifty absolute, so that, although it is quite possible to suffer from facultative Hm. and pass through youth without any symptoms of eye-strain, sooner or later they will appear. Good health, plenty of outdoor exercise, and not too much application to books,

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will ward off eye-strain for a long time, but in these days of examinations the day must surely come when the young student must "cram," when he must read four or five hours a day by artificial light, when he must put in more work and take less play—in other words, when he must use his muscles of accommodation for a much longer time. And now, after a few hours' reading, his head aches, his eyes pain, and the type runs together. Of course many such cases may occur from simple overwork in emmetropes; but I am confident that many a young man has broken down reading for his "Tripos," simply because he was hyperopic and had over-strained his eyes. I have known several men suffer from eye-strain for the first time through taking up German or Hebrew; the fine strokes that have to be recognised in order to distinguish the different letters (especially is this so in Hebrew) put an extra strain on the accommodation; and if the eye starts with a deficit, as it does in hyperopia, eye-strain is sure to ensue, and we get all its attendant symptoms.

If symptoms of eye-strain occur amongst the upper classes who suffer from this low form of hyperopia, how much more must they occur in those who spend their lives at close work in badly lit and badly ventilated rooms, with little or no outdoor exercise, and often insufficient food, and yet the large army of seamstresses and compositors, etc., who find their way to the out-patient room of an ophthalmic hospital, are, I believe, only a small fraction of the number that really want relief but do not recognise it, because they see well without glasses. Those who do come for advice generally tell the same tale; they are at work, say, with the needle from eight in the morning till eight or ten at night, and towards evening they complain that their vision becomes less acute—it is difficult to thread a needle, and their eyes and head ache. It is but natural; their ciliary muscles have been working at high pressure all day, and in their little way have done as much work as the leg muscles would in a thirty-mile walk. Surely in an emmetropic eye we should expect fatigue under such circumstances; how much more, then, in hyperopia?

With advancing years the latent hyperopia becomes gradually (and finally, about the age of forty, entirely) manifest, and with diminution of accommodation range the hyperope necessarily becomes prematurely "presbyopic." As we should expect, symptoms of eye-strain are much more common in presbyopes who are hyperopic than in those who are emmetropic or myopic.

One of the results of eye-strain in young hyperopes, or in those who have to make great efforts to see small objects, as watchmakers, etc., is spasm of the ciliary muscle, whereby vision is accommodated to near objects and the patient rendered artificially myopic. This spasm is usually accompanied by contracted pupil from associated spasm of the sphincter of the iris, both conditions being caused by direct or indirect irritation of the third nerve.

Hyperopic headache is often accompanied by twitchings of the eyelids.

To recapitulate, then, besides the many symptoms of asthenopia already described, there are special indications of hyperopia, which are—

1. Spasm of ciliary muscle, producing apparent myopia.

2. Sudden failure of the ciliary muscle from fatigue causing obscurations of vision.

3. Convergent strabismus, and in addition

4. Apparent divergent strabismus.

The angle γ is larger, *i. e.* the distance between the macula and optic axis is increased, which causes the axis of the cornea to appear to diverge.

There are also certain physical signs noticeable in hyperopes.

The eye is flatter than normal, and often markedly smaller; if the eyeball be turned strongly in or out the equatorial region presents a much sharper curve than in the normal or myopic eye, showing its shortened axis.

The cornea is often smaller than normal. The face is sometimes flat-looking, and the two sides assymmetrical.

The ciliary muscle of a hyperope is always larger than normal; this is specially marked in the annular muscle of Müller, and is due to excessive use.

THE DIAGNOSIS OF HYPERTROPIA BY EXAMINATION.—1. Vision is improved, or is as good, with convex glasses.

2. Retinoscopy gives a shadow moving "with" with a plane mirror, and a reverse shadow with a concave mirror, and the brighter the shadow and the quicker its movement the lower the H.

3. The indirect ophthalmoscopic examination

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shows an image of the disc larger than normal, and diminishing on withdrawing the lens from the eye.

4. In the direct ophthalmoscopic examination, if the observer's accommodation is relaxed a convex glass is required in the ophthalmoscope to obtain a clear image of the fundus. If the H. be high, on removing the ophthalmoscope from the eye an erect image of the fundus is seen moving with the observer.

TREATMENT.—If the patient is under fifteen use atropine, whenever possible, to paralyse the ciliary muscle, and over fifteen up to forty use hom-atropine. Over forty no mydriatic is necessary, and may even be dangerous, as at this age there may be a tendency to glaucoma, which is especially determined in hyperopic eyes because of their formation (a small flat cornea and large ciliary muscle tending to block the filtration angle). By means of retinoscopy and testing before the type we ascertain the atropine correction of the defect.

We must bear in mind that as the accommodation is never so relaxed as when it is under a mydriatic, we must forbear to give the full atropine correction. One good rule is to neutralise the whole of the Hm. and one quarter of the Hl. Thus a patient with Ht. = 2·5 D., of which ·5 D. is manifest, and consequently 2 D. latent, should have as glasses to wear always + 1 D., *i.e.* $\cdot 5 + \frac{1}{4} \times 2$.

As a matter of fact, the ideal treatment would be to correct the whole of the hyperopia, because we thus render our patient emmetropic, and restore the harmony between the accommodation and convergence.

The correct treatment, I am sure, is to decide each case on its own merits, the degree of H. and the age of the patient being very important factors.

In patients under twelve give the full correction less 1 D. if the H. is of medium amount, say up to 4 or 5 D. The patient will very often at first complain of the glasses, but, by the rigid use of them, taking care that they shall be circular, so that he cannot look over them, the eyes will soon conform to the altered conditions, and the patient will probably return in a couple of months perfectly satisfied.

If the H. be higher it is as well at first to take off from the atropine correction a little more than 1 D., viz. 1·5 or 2 D., but in young people not more.

This plan, when adopted with those that have developed internal strabismus, has often the happiest results, for we may cure the squint without resorting to an operation, and the effect is in direct ratio to the youth of the patients. Moreover, these are the hyperopes who can most readily be made practically "emmetropic," and therefore get the greatest gain from the glasses, for their very defect, the squint, shows that they have been unable to dissociate their accommodation and convergence. It is this habit of using their accommodation in excess which prevents many hyperopes from taking full correction. They cannot unlearn the habit, and naturally the older the patient the more probability is there of this being the case. In such cases we must commence with a much weaker convex glass, and we must hope to be able to increase it later.

We must treat every case on its own merits; when the amplitude of accommodation is great, the patient will probably prefer a weaker glass than we wish to give, and *vice versâ*. When the hyperopia is slight, the vision good, and no symptoms of strain are present, we should abstain from ordering any glass.

In advanced youth and early middle age it is unwise to follow any rule about the deduction for atropine, because the personal equation is a most important factor. Wait for the patient to recover from the mydriatic, and then examine before the type, taking off the smallest amount that will just give distinct vision for distance. For instance, supposing 6 D. represents the H. The patient may only stand 3 D., and ·5 added produces blurring, therefore order + 3 D., and in a few months, when the eye has adapted itself to the altered circumstances, you may be able to order + 4 D. If you insist on the higher glass at first, the patient finds he cannot see so well with the glass as without it, and there is the danger that he will not use his correction.

Sometimes these patients will receive great benefit from their partial correction when not using the eyes much for near vision, but will complain that they feel they want stronger glasses for near work; these you are always justified in ordering under the circumstances, and the strength may be up to the full atropine correction.

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In later life, when the latent H. has become manifest and absolute, two pairs of glasses are required, because the distant and near vision must both be corrected. For distant vision give the strongest glass the patient will accept, and for near vision ascertain the correct glass by the rules for the treatment of presbyopia.

To avoid the constant change of glasses, bifocal lenses or Franklin glasses are much to be recommended. The patient looks through the top of the glass for distance and the lower part for near work. Or the patient may have his distant correction put into spectacles, and for reading he may place in front of these pince-nez, to correct his presbyopia. If Franklin glasses are ordered the patient should be warned to lift the glasses when going up and down steps and stairs.

It is important to remember that hyperopia tends to decrease towards emmetropia. A child may be hyperopic to the extent of 2 D., and as the growth and development of the different parts of the body proceeds the flatness of the eye may disappear, and by puberty it may be emmetropic. For this reason it is necessary to re-examine the eyes of children every year, in order to make quite sure that they are not wearing a too strong convex glass, which would induce an artificial myopia, which, in turn, might lead to a real myopia.

This tendency for the eye to grow normal is often interfered with by the presence of asthenopia, hence one of the benefits derived from glasses in young children is the increased possibility of abandoning spectacles.

Some Cases of Snake-bite treated with Calmette's Serum.—A. Boddaert cites several cases in which persons bitten by snakes were successfully treated by Calmette's serum injected subcutaneously. The author has no new cases to report, but desires to call the attention of physicians to a form of treatment which is as simple as it is efficacious, and to impress upon colonial government officers the necessity for the greater diffusion of a method which in British India alone would save the lives of a large number of persons annually.—*Medical Record*, March 29th, 1902.

THE DOSAGE OF CHLOROFORM.*

(A critical review of prevailing methods from a clinical standpoint.)

By WALTER TYRRELL, M.R.C.S., L.R.C.P.,

President of the Society of Anæsthetists; Senior Anæsthetist to St. Thomas's Hospital; Anæsthetist to the French Hospital.

AFTER the publication of the report of the anæsthetic committee of the British Medical Association, there appeared a paper by Dr. Waller in the 'British Medical Journal' criticising the report, and dealing with the dosage of chloroform.

On a question of dosage one naturally turns to the British pharmacopœia. I found it there stated that the dose was 3 to 10 minims, but no instruction as to inhalation is given. Turning to other potent drugs, I found that the dose of tincture of aconite is given as 5 to 15 minims. I know some physicians prefer to give 1 minim every hour until a certain effect is produced, but I doubt if there is any one who would care to commence with the full dose of 15 minims. Yet this is just the difference between the gradual method of the administration of chloroform and the sudden or rapid method of administering as strong a vapour as can be inhaled. A certain amount of latitude in dosage is necessary because when a patient is under the influence of a drug it requires a much smaller dose to keep that influence up. When a man is drunk, I imagine it takes very small doses to keep him in that condition, but the whole world is familiar with the fact that the dose of alcohol to produce intoxication varies enormously in different individuals. Is it not the same with chloroform? Have not fatal cases been recorded in which remarkably small amounts of chloroform had been used, and others in which a satisfactory anæsthesia has only been maintained by the use of enormous quantities such as would certainly prove fatal to the average person?

If the human body were a machine which worked with mathematical accuracy, we might doubtless arrange various volumetric solutions of chloroform suitable for different ages and sizes, and the anæsthetist would only be required to be a competent dispenser of drugs. The idea of a volumetric solution is not a new one. In 1849 Snow employed a bag containing an atmosphere

* Address delivered before the Society of Anæsthetists.

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with 4 per cent. of chloroform vapour in it, and it must be nearly thirty years ago that this method was revived by Mr. Clover, and for some years it was used in many of the London hospitals. It was soon found, however, that a solution of chloroform suitable to one person was deadly to another, and that dangerous complications were apt to occur before the patient was fully under its influence, and that when the patient was once anæsthetised the administration had to be stopped and commenced again on signs of returning consciousness approaching. In other words, it was found that the solution of chloroform necessary for the desired narcosis was dangerous in many cases before that narcosis had been attained, by reason of its producing respiratory and circulatory and cardiac complications, and was dangerous after the production of narcosis by reason of its producing chloroform toxæmia unless given very intermittently, and therefore other methods were had recourse to by which chloroform could be commenced by administering a weak solution, and that solution slowly and gradually strengthened until the desired narcosis was produced, when the strength of vapour could be again more or less slowly diminished, and so keep the narcosis at the desired level. To do this successfully the chloroformist must not only have full control over the strength of vapour he is administering, and be able to increase or diminish it at will, but he must be able to interpret the various signs exhibited by the patient as to whether they indicate an increase or decrease in the strength of the vapour. Signs of danger are more frequent before complete narcosis is produced than afterwards. Only some of these are to be treated by increasing the strength of the vapour, while others necessitate the slowing down of the administration.

If we used volumetric solutions of chloroform we should require to have at hand four or five different strengths to work with, and even then by far the most important factor in the safe administration is the experience which has been acquired by the administrator. The latter statement, which is one of the conclusions of the Anæsthetics Committee of the British Medical Association, has been much criticised, but does it not apply to almost every department of medicine and surgery, and especially to surgery? It is true that with anæsthetics a question of life or death is involved, and so it is with a large number of major surgical operations. Take, for instance, the surgical treatment of a case of intussusception in an infant, in which dexterity and rapidity are important factors. A surgeon in the remotest parts may be called upon to undertake such a case, but would it be fair to compare the statistics of a remote village cottage hospital with those of a large hospital in a populous town? Yet the questions of life or death are probably more involved in the surgical procedure than in the administration of the anæsthetic. Our critics further tell us "that the practical administration of anæsthetics still remains before the profession as a problem inadequately met. Its data remain still insufficiently determined. It is admitted that the surgical narcosis required of us leaves between it and death a margin not too wide, and we are told that the difference seems almost one of quantity rather than quality." This is perfectly true, but it is impossible to fix that quantity even with a considerable margin, because the other side of the equation is not a mathematical unit. When commencing to give chloroform to a patient it is not possible to say how much will be required to produce surgical narcosis. We know roughly that a vigorous, powerful man in good health will require a great deal more of the anæsthetic to produce the desired effect than the more feeble individual, and speaking of chloroform I think we are agreed that in the vigorous, powerful, and healthy individual the margin between surgical narcosis and death is not so wide as in the more feeble, and that this is due to the fact that the larger dose for the former necessarily brings him nearer to chloroform toxæmia than the smaller dose, which is sufficient to anæsthetise the latter. On this account ether is often chosen for the vigorous and stalwart subject, although for some reasons chloroform would be the most suitable, and on this account also in cases where chloroform is an absolute necessity, we commence the administration with ether so as to avoid nearing the margin of safety with the former drug. Moreover, the same patient will often require a much larger dose at one time than at another. The specialist is sometimes asked to give chloroform to a patient for a secondary operation because some weeks previously that patient had exhibited signs of danger when an anæsthetist of small experience had been the administrator. In the meantime that patient had

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been five or six weeks in bed suffering from fever, and the specialist is able to produce surgical narcosis with the greatest ease and safety, probably requiring only half the amount of the drug used on the former occasion. Experience is probably more necessary for robust patients than for feeble ones, chiefly for the reason that the larger dose required brings the patient nearer to the margin of toxæmia, and also because the stronger vapour necessary is much more apt to produce complications of a dangerous nature, even before narcosis has been produced.

These are the main reasons why no single volumetric solution has been found to meet the requirements of the anæsthetist in producing surgical narcosis. A critic of our methods truly says "that such narcosis entails depressing the activities of the organism toward a depth not far above that where they wholly cease, and often retaining them so depressed for periods relatively long. It seems essential for safety that the manner of descent be gradual and smooth, the entrance on that vital plane of lower level quiet, so that the tenour of the afterway along it may be steady and secure from oscillations. Every descent below the requisite level must be fraught with danger, and every reascent require a fresh plunge, and, consequently, a new adjustment difficult to make." This gradual and smooth descent, which is undoubtedly so essential for safety and the maintenance of a level of anæsthesia steady and secure from oscillations, is just what has been found to be unattainable by any one volumetric solution. I think anæsthetists are agreed that the solution of chloroform must vary in strength according to the requirements of the individual being operated on, and that full control over the strength of the vapour must be maintained by the administrator. To attain this end, the drop bottle, capable of dropping single drops, and Junker's apparatus, with its various improvements and modifications, are now very generally used, at any rate by those who have received their medical education in London. We know that no method of administration of chloroform is free from danger, but we believe that this method is made relatively safe through the power it gives us of varying at will and fully controlling the strength of the vapour used.

There has always been a difference of opinion amongst leading authorities as to the strength of vapour which should be used to commence with, some holding the view that it is best and safest to give as strong a vapour as can be freely inhaled until signs of narcosis appear and then slacken off to a weaker vapour, and so maintain a level of anæsthesia, while others declare, and the latter include, I believe, the whole of the London teachers of anæsthetics, that this method entails serious risk, both from overdose and from respiratory complications caused by the irritating effect of the vapour. Personally, I have not the shadow of a doubt as to the greater risk of commencing with a vapour of full strength, although it may be necessary to attain that strength before surgical narcosis is accomplished. At the same time there seems to be a consensus of opinion amongst those who advocate graduating the strength of the vapour that a too slow administration, and therefore a too much graduating, has also its dangers and drawbacks. This is undoubtedly true, but the greater question, especially for the beginner, is: How do the dangers of the comparatively slow administration compare with the dangers of the more rapid but still graduated method? In the first place, I think all anæsthetists of experience believe in chloroform idiosyncrasy, a peculiar susceptibility to toxæmia from very small doses. In the slower administration the danger-signal is given before positive danger has arisen, and by prompt action serious danger is altogether avoided. The more rapid the administration the more likely is excitement, struggling, and holding of the breath to occur, and the greater is the risk of too strong a vapour being locked in with the residual air of the lungs in these peculiarly susceptible subjects.

The only danger which is worth considering which is actually due to the slower administration is the nausea and vomiting coming on before surgical narcosis has been produced, and which is sometimes accompanied by a more or less alarming degree of syncope. Every chloroformist is familiar with these same phenomena occurring some time after the administration has been stopped when the patient is emerging from his anæsthesia. Then it is known to be unavoidable, and appropriate treatment invariably brings about a good recovery. Such syncope due to nausea and impending vomiting does not occur with anything like the same frequency during the process of anæsthetisation as when emerging from that condition, but when it

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does occur just as an operation is about to be commenced, the surgeon thinks it is due to a want of skill in the administrator, while all concerned cannot but feel that an attack of syncope is a bad preparation for the strain of a prolonged narcosis. The experienced administrator may often prevent such an attack by cautiously pressing the chloroform when such signs as swallowing, faint attempts at retching come on with pallor and shallow respiration. But it is not always possible to avoid vomiting, and the greater the degree of syncope accompanying this condition the longer the delay before such vomiting occurs. With it, however, comes a rapid return of colour to the face, the respiration becomes normal, and the pulse often better than before the inhalation commenced, thus showing that the syncope is due to other causes than a primary cardiac one or secondary to respiratory difficulties. Seeing the amount of fluid sometimes ejected from a stomach presumed to be empty, I have wondered whether this vomiting was caused by that organ eliminating the drug as is, in the opinion of some recent investigators, the case with morphia. At the same time, one has frequently seen symptoms of a very similar character produced through the nervous system quite independent of the inhalation of chloroform or the ingestion of morphia or other drugs. I remember seeing identical symptoms in a strong man who was being vacinnated prior to proceeding to South Africa on a big game shooting expedition. I have never seen this syncope and vomiting occur before narcosis has been produced in the case of anæsthetising a child who is already asleep, which is necessarily a very slow process, and although I have sometimes experienced it in children, I do not recollect having seen it in children who scream during the administration, notwithstanding that the inhalation is commenced very gradually. Most authorities agree, however, that this danger and drawback may be avoided by a less sparing use of the drug. At the same time I think the risk it incurs is small by comparison with the risk of overdose by more rapid administration in a peculiarly susceptible subject. The experienced anæsthetist is often able to steer a clear course through his greater ability to gauge his patient. I am therefore in the habit of teaching that the strength of vapour should be very weak to begin with, and slowly increased until narcosis is produced, when, in turn again, the strength of vapour is diminished to keep the anæsthesia at the proper level. If from intermittent use of the drug this level is not kept up, we may get syncope from impending vomiting, as at the end of an administration, when the patient is emerging from his anæsthesia, or we may get syncope due to the surgical procedure, both of which conditions occurring in the middle of an operation are of much more serious import, and can only be avoided by a complete control of the strength of vapour used. No one volumetric solution could give us that control which it is absolutely necessary to possess.

As I have already said, leading authorities differ as to the strength of vapour which should be used to commence with. We have to confess that a large measure of success attends the Scotch method of pouring a couple of drachms, more or less, upon a towel, and being guided entirely by the effect of the resulting vapour upon the patient. We have, however, the authority of Dr. Conan Doyle that matters do not always go smoothly. In his story 'His First Operation,' the student who fainted when the surgeon examined the scalpel found when he recovered that there had been no operation as the patient could not stand the chloroform. I believe there is not always a public inquiry into cases of death under anæsthetics north of the Tweed, and it is fair to assume that such cases are not reported with the same regularity as in England.

A correspondent at Glasgow writes to me, "One never sees now chloroform given by the old Scotch method of pouring a couple of drachms or an unknown quantity of chloroform into a conically folded towel. It is given by drops on a single layer of towel. A member of the surgical staff of the Glasgow Western Infirmary told me that chloroform given gradually on a towel was the one anæsthetic used, and on my asking why ether was never used, he said he thought tradition had more to do with it than anything else."

The most recent publication on 'Chloroform, that of Surgeon-Lieut.-Colonel Laurie, of Hyderabad Chloroform Commission fame, also advocates the method of commencing with a vapour as strong as can be inhaled without distress or choking, and states that if this is attended to the more rapidly the chloroform is given the better, till anæsthesia is produced. To this end he uses

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a cloth cone or cap, with a little absorbent cotton stitched into the apex. A drachm of chloroform is poured on to the cotton in the cap every three quarters of a minute or minute, and the cap should be held loosely but closely over the mouth and nose. Although Dr. Laurie commences with a very strong vapour, he insists that the chloroform must be adapted to the patient, not the patient to the chloroform. His statistics, which give one death in a continuous series of 17,300 cases, it must be confessed show a very remarkable result. He says that out of 14,174 administrations during 1892 to 1899, of which complete notes were recorded, there were 302 cases in which the breathing became obstructed, and in 18 of these cases artificial respiration was considered necessary, and in the remainder, where there was merely some embarrassment of the breathing, relief was afforded by pushing forward the lower jaw. These statistics seem to accentuate the conclusions of the Anæsthetic Committee, that no method of administration is free from danger, and that the occurrence of danger depends largely upon the administrator who employs any particular method. I should have mentioned that in Dr. Laurie's series the one fatal case occurred during his temporary absence from Hyderabad.

Dr. Laurie admittedly uses a strength of vapour which he knows would be fatal if continued for a minute after anæsthesia has been produced. His two great safeguards are: (1) That while producing anæsthesia he removes the chloroform entirely during any hesitancy in breathing, and especially during struggling and holding of the breath, and so avoids the inhalation of a specially toxic dose, which may accumulate in the facepiece in a very few seconds in all methods where a drachm or more of chloroform is poured on at a time. I need scarcely say that this danger is clearly recognised by all administrators, whether they hold the views of the Hyderabad Commission or not; (2) he stops the administration directly anæsthesia is produced, and does not continue it until some sign of returning consciousness occurs.

What strikes one first on looking over his notes of cases is the astonishing rapidity with which some of his cases were anæsthetised, and the length of time which elapsed after that point had been arrived at before more chloroform was required.

In Case 1 a drachm of chloroform was used for a child æt. 4 years; a lachrymal probe was passed after 35 seconds' inhalation, the operation being concluded in 50 secs. This, however, is called partial anæsthesia.

In Case 2 a boy æt. 7 years was fully anæsthetised within a space of 2 mins.; 2 drachms of chloroform were employed, and the operation of lateral lithotomy, taking 8 mins. to perform, was accomplished without the inhalation of any more chloroform after anæsthesia had once been established.

In Case 4, male, æt. 56, the production of anæsthesia occupied 5 mins. 27 secs., but the patient, owing to respiratory irregularities, was only inhaling chloroform for 4 mins. 5 secs. Five drachms of chloroform were employed to produce anæsthesia, and the operation of dilating a stricture of the urethra was accomplished with only two further administrations of chloroform amounting to 2 drachms, making 7 drachms in all administered in 16 mins. 20 secs.

To mention only one more case, No. 7, chloroform was given in 1 drachm doses, at first for 1 min., then air alone for 6 secs., then chloroform for 8 secs., then air alone for 42 secs., then chloroform 19 secs., then air alone 22 secs., chloroform again 38 secs., then air alone 24 secs., at the end of which time, viz. the last 24 secs. of air alone, the patient became anæsthetised. This patient was a male æt. 43 years. The time occupied to produce full anæsthesia was 3 mins. 39 secs., but during this time the patient inhaled air alone for 1 min. 37 secs. We are told that this 1 min. 37 secs. of air alone saved the patient from overdosage. No one will hesitate for a moment to accept this view, for if that important minute and odd seconds did not save the patient from overdosage, what did prevent it?

When a drachm of chloroform is poured into a cap with a little absorbent cotton stitched into the apex, and such dose repeated every 45 to 60 secs., it must be clear that if the cap is not removed during holding of the breath, even for a very few seconds, a most toxic vapour would accumulate under the patient's nose ready for inhalation immediately the breathing recommenced, and overdosage would certainly result. The avoidance of a toxic dose is the aim of every chloroformist, and does not entail an acceptance of the Hyderabad or any other views of the

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physiological action of chloroform. If I were obliged to give chloroform in the rapidly repeated drachm doses recommended by Dr. Laurie, I should hail natural effort to prevent overdose by holding of the breath as my one safeguard. To disregard that safeguard by not removing the inhaler, but allowing a vapour to accumulate ready for inhalation when breathing should recommence, the strength of which vapour all the world will allow might be toxic, would not only be to court disaster, but would be an outrage on common sense. I cannot but think that this method, even in Dr. Laurie's able hands, would be found more dangerous in this climate than in the climate of India. It is certainly in marked contrast to the absolutely smooth and regular breathing of a sleeping child being slowly narcotised with chloroform.

To conclude, I think the day is far distant when the physician will sit at his table and write a formula for each patient for the anæsthetist to dispense, and when that day does come, I have no doubt that a machine will be invented to carry out the mechanical process.

B. K. RACHFORD ('Medical News') reports four cases of acute influenzal nephritis occurring, not as a sequel, but as a part of the influenzal attack. The kidney symptoms in every instance occurred while other active influenzal symptoms were still present. The influenzal poison attacked the kidneys and produced a most violent acute nephritis, just as it, in other cases, attacks and produces inflammation of the lungs, the intestines, the meninges, and other parts of the body. In these cases it came on more quickly and violently than nephritis produced by scarlatina, diphtheria, and the other acute infections. In the author's experience the worst symptoms occur, as a rule, within six or seven days after the kidney is attacked, and if complete suppression and profound uræmia do not destroy the life of the child within the first week of the disease, a sure and steady improvement begins which leads to complete recovery. It is possible that in some of the cases a chronic nephritis may be established. In adults the disease is very different. The acute hæmorrhagic type occurring during the influenzal attack is nothing like as common as it is in children. Chronic nephritis, however, resulting from repeated attacks of influenza, is much more common in the adult than in the child.—*Philadelphia Med. Journ.*, March 22nd, 1902.

Amœbic Dysentery in Children.—Samuel Amberg ('Bull. of Johns Hopkins Hospital,' Dec. 1901) reports five cases of this infrequent disease. The first factor of interest is their grouping; in two instances the disease befell members of the same family exposed to the same influences. A third child of their company was taken sick with the same symptoms. The clinical type to which the cases belong is one of moderate intensity, in which the appetite and general health are good, with no fever or acceleration of pulse worth mentioning, but with stools varying from two to six in twenty-four hours. One of the cases was more severe, general nutrition being decidedly interfered with, the pulse being increased in frequency, the temperature rising irregularly, particularly at night, and the stools varying from eight to fifteen in twenty-four hours. Pain was not complained of except in this special case. As to complications, in two instances there was prolapsus ani, which was reduced spontaneously. There was no sign of affection of the liver in any case. The reaction of the fæces was mostly alkaline, seldom acid. Sometimes in the acid stools the amœbæ continued to move for from two to three hours. In some cases the fæces were firm and formed, with bloody mucoid masses on the surface. Sometimes a formed stool was passed followed by blood, either liquid or, rarely, in clots, accompanied by more or less mucus. In other instances the passages were semi-solid or uniformly liquid, of different colour, with mucoid masses and blood intermingled. The odour of the fæces was always very offensive. In three cases there appeared toward recovery, in the place of the mucoid masses, stools of peculiar gelatinous consistence, which were found to be composed of continuous layers of epithelial cells. The diagnosis was based on the finding of motile amœbæ containing red blood-corpuscles. The blood was examined in these cases. In four there was a varying degree of anæmia, which found its expression more in a deficiency in hæmoglobin than in the red blood-corpuscles. In all the cases there was a leucocytosis, in most of the counts not a very high one. Where the leucocytosis was more pronounced the number of polynuclear neutrophile elements was increased. As to eosinophiles, the number of blood examinations does not allow of absolute conclusions. There was distinct although slight eosinophilia in one case only.—*American Journal of Obstetrics*, March, 1902.

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A CLINICAL LECTURE

ON

FOUR CASES OF PERFORATING GASTRIC ULCER;

TOGETHER WITH NOTES OF A CASE OF ACUTE DILATATION OF THE STOMACH.

Delivered at St. George's Hospital, March 4th, 1902.

By G. R. TURNER, F.R.C.S.,
Surgeon to the Hospital.

GENTLEMEN,—The subject which I wish to bring before your notice this afternoon is one which is of immense importance, because on how soon the cases of it are treated depends very largely the ultimate result. It is the subject of perforations of gastric ulcers, of which I have had several under my care more or less recently, and which you may have had an opportunity of seeing in the wards. First of all, I will read you the notes of one of the cases which is now in the Princess Ward, and which is luckily doing well. I believe the case is on the high road to recovery. It was an acute case of gastric ulcer which had perforated. You can divide the cases of gastric ulcer which perforate into acute and chronic, and the first cases I shall mention are two cases of acute gastric ulcer with perforation, and later I shall deal with some of the more chronic variety which have been under my care in recent years. I shall only select the cases which have been here during the last two or three years, and therefore they will be cases which probably most of you have had an opportunity of seeing.

The case which I want first of all to draw your attention to is that of a young girl æt. 17, who is now in No. 5 bed in Princess Ward. Her age is about that at which you meet with gastric ulcer in women. Curiously enough gastric ulcers in women are very much more common than in men, and the time of their appearance and of their perfora-

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tion varies in the two sexes. If you look at a number of cases of perforating gastric ulcer you will notice that they occur much earlier in women than in men. I think I am right in saying that in men gastric ulcers occur and perforate more between the ages of twenty-five and forty than at any other time of life, the age in women being fifteen to twenty-five. Well, our first patient was a girl æt. 17, who had had pain and vomiting half an hour after food since Christmas, 1900, that is to say, for fifteen months. She had never had hæmatemesis or melæna. As you know, gastric ulcer is constantly accompanied by vomiting of blood, and all such cases of hæmatemesis are accompanied by melæna. There are some cases, however, in which melæna only is present; cases in which there has never been any hæmatemesis, that is to say, cases in which the effusion of blood has been sufficient to stain the fæces, but has not been vomited. It is said that 11 per cent. to 13 per cent. of cases show melæna but have no hæmatemesis; but wherever there is hæmatemesis in this condition you will be sure to have melæna also. This patient had neither melæna nor hæmatemesis. She had what she called a bilious attack soon after Christmas. Her bowels had always been constipated. On February 15th last, after a small meal, she had sudden acute abdominal pain in the left hypochrondrium, and vomited several times. The bowels acted after this attack. The pains continued and the vomiting ceased, though the attempts at vomiting persisted. I want you to notice that feature of retching continuing after vomiting had ceased; it is rather a characteristic feature. In cases of acute perforation of the stomach it stands to reason that when there is an attempt at vomiting the contents of the stomach are not expelled by the mouth but are expelled into the peritoneal cavity. Thus you may get a feeling of sickness without any actual vomiting. When she came in she had a pulse of 140, that rapid pulse that shows some peritoneal trouble. Her temperature was 103°, that is to say, she had recovered from the temperature of collapse. She had been collapsed first of all, and, as you know, when people are collapsed their temperature falls, even to subnormal. She was very anæmic, and anæmia is very frequently present in these cases of gastric ulcer. I asked a physician the other day what was the cause of gastric ulcer, and he said he did not know, but in nearly all cases anæmia was present. That also is the experience of surgeons. On examining her abdomen we found it not very much distended. There was great tenderness all over the belly, especially in the left hypochrondrium. There was no very marked rigidity, and it moved slightly on respiration. The liver dulness was present, and there was a splashing sound heard. When I saw her I concluded she was suffering from perforation of gastric ulcer, and accordingly immediate operation was advised. I opened the abdomen in the middle line above the umbilicus, and when I got through the peritoneum there was a quantity of fluid present, which came out through my incision. On the anterior surface of the stomach, near the pylorus, there were obvious marks of old gastric ulceration and cicatricial contraction, not sufficient to cause obstruction of the pylorus, but it was very obvious she had an old ulceration there. That, however, was not the seat of her present trouble. On the extreme left end of the stomach, which was adherent to the colon, some adhesions, which were evidently newly formed, were seen and separated; and when I got through the great omentum and worked my way up on the posterior part of the stomach, quite close to the entrance of the œsophagus, that is to say, the cardiac end, there was found to be a very deeply seated perforation quite close to the abdominal aorta, where the stomach and œsophagus join. Before I tore through the great omentum to get to the seat of perforation, I carefully examined the anterior surface of the stomach, but there was no evidence of perforation there at all. You know that perforations of the stomach are very much more common on the anterior than on the posterior surface for this reason: that although ulcerations of the stomach are more common posteriorly than anteriorly, the stomach posteriorly rests on certain structures, to which it forms adhesions, such as the pancreas. Here is a specimen of that, a large gastric ulcer being adherent to the pancreas. So it comes about that when ulcers perforate, if there is leakage of an ulcer posteriorly, the ulcer is often sealed up by the non-movable structures such as the duodenum and pancreas, which rest in contact with the posterior surface of the stomach. But on the other hand, when an ulcer perforates anteriorly there is nothing except the abdominal parietes, which are constantly moving with respiration, to

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arrest the flow of the contents, and so an anterior perforation is much more common than a perforation on the posterior aspect. In this case, then, I found a perforation on the posterior surface of the stomach, quite close to the abdominal aorta, very deeply situated. The depth of the situation of this perforation and its important anatomical relations led me not to attempt any suture. When perforations are situated posteriorly on the stomach —and it is only in about 2 per cent. of the cases that they are so situated,—the surgeon will constantly find that he must not attempt to suture, and, indeed, he may find it physically impossible to do so. He must content himself with doing what I did in this case, that is to say, packing the parts around with strips of gauze, thoroughly draining the peritoneal cavity and flushing it out, so that no portion of food or other gastric contents are left behind. That is what I did in this case. I saw from the quantity of fluid which had already escaped that it was certain there was not only fluid in the upper part of the abdomen, but there was a collection of it in Douglas's pouch below. So I made another incision just above the pubes, and washed out Douglas's pouch with simple warm water. The pouch was full of stomach contents. I drained above the pubes by means of a glass tube, and drained the flanks by means of a couple of large india-rubber tubes. I made my incision go nearly to the xiphoid appendix. In addition I had four strips of gauze packed down to the seat of the ulcer, which I brought out of the wound in the position shown in this diagram. In that way I shut off the perforation from the general peritoneal cavity by gauze packing. I flushed out the peritoneal cavity until the water came away absolutely clear. Then I stitched up the two wounds in the ordinary way. You must remember that the ulcer was so deep that I could not suture it, and that the contents had been diffused throughout the abdominal cavity. When there is perforation of the posterior part of the stomach you will have effusion of the stomach contents into this lesser sac of the peritoneum, and if the foramen of Winslow is still patent the contents will come out of the foramen into the general peritoneal cavity, and there is no doubt this happened in this case. The foramen of Winslow was open; but sometimes in these cases that foramen is blocked by adhesions or is stuffed up by coils of intestine, and instead of having diffusion of stomach contents all over the cavity you will have a form of local infection about which I shall speak presently. In this case there had been a posterior perforation, the effusion of the stomach contents into the lesser sac travelling out through the foramen of Winslow, so that when I opened the abdomen I was at once met with the stomach contents, and they had come down into Douglas's pouch. In such cases you have to drain this part of the peritoneum just as much as you have the upper parts. As far as her subsequent history is concerned, the progress so far has been all that we could wish. I took out the pieces of gauze on about the fourth day, having shortened them each day. I took the glass tube out of Douglas's pouch about the third day, and she has, inserted into the wounds in the flanks, very small tubes instead of the large ones at first employed. There is still a free discharge of non-smelling healthy purulent matter from the neighbourhood of the stomach, and now she is having some food by the mouth, though, of course, nothing as yet except milk and beef tea. Seeing that it is a fortnight since the operation was done, I think she will go on to complete recovery. Her pulse rate throughout the whole of the time was rapid, 140; even to-day there is a note that it is 96. It has been as slow as 80, and that rapidity of pulse is likely to continue until she gets perfectly well. Still, other things being equal, the chances are enormous that this will be a successful case of operation for perforating gastric ulcer.

There is another case of the acute kind which I should like to read to you. That was a case which was under my care in November. It was that of a girl æt. 24, a shop assistant at a confectioner's. She was engaged in arranging some bottles of sweets, when a mouse jumped out and frightened her. She gave a sudden start, and that was probably the cause of the giving way of the ulcer, which probably had existed for some time. She had a sudden acute pain in the abdomen followed by vomiting, which continued but slightly. Here, also, the vomiting was not profuse, for the reason I have given, that when there is perforation you do not get profuse vomiting, because the contents of the stomach go into the peritoneal cavity. She was very ill, there was much pain and tenderness in the abdomen, which was distended, quite rigid, and resonant, and the liver dulness was absent.

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You know in these cases when fluid and gas leave the stomach and enter the general peritoneal cavity, the gas has a tendency to rise to the topmost part of the abdomen and to get in front of the liver, so as to make the liver dulness less than it should be. That symptom of absence of liver dulness in the earliest stages is of the highest value. Perhaps in later stages, when there may be peritonitis, and the transverse colon is very much distended, this absence of liver dulness is not so important a sign; but if, as I say, you have absence of liver dulness in the early stage, with resonance shifting according to the position of the patient, that will very likely indicate the presence of air or gas in the abdominal cavity. In this case, then, there was absence of liver dulness. I operated upon this patient, performing a median laparotomy above the umbilicus, and found a perforation in the stomach on the anterior surface, about the greater curvature towards the left side. It is unusual to have perforation there; perforations are much more common about the lesser curvature. It was recent in origin, and obviously there had been a little adherent omentum, and probably it was when she was startled that this adhesion of the omentum to the perforation gave way. There was much lymph and fluid, and there were signs of commencing peritonitis. As it was on the anterior surface I was able to suture it by three rows of Lembert stitches. I irrigated the abdomen with saline fluid, and left a pint of the saline fluid inside. That is a way of treating the shock which is so often present. When you have irrigated the abdomen you need not trouble about washing all the clear fluid away. Leave, if you like, a little saline fluid in the abdomen; by so doing you are probably benefiting the patient. In this case another opening was made below the umbilicus and a drainage-tube inserted above the pubes. I was able to suture the ulcer, and I did it by means of the Lembert sutures. With reference to the suture of gastric ulcers, the main thing is to suture them quickly. If you take up all the coats in so doing, I do not think it matters very much; in fact, I think it is the right thing to do if the fluid is gushing out of the stomach. You had better put the suture through all the coats, and thus sew up all the coats together, and you can afterwards go in for the refinements of suturing, such as sero-muscular coat to sero-muscular coat, but do not waste time by going in for elaborate suturing first. This patient made a good recovery, and is perfectly well. Her convalescence was slightly retarded by a tenderness and œdema of the calf of one of her legs fourteen days after the operation. I have never been able to make out whether she has had thrombosis or phlebitis, and it did not give her much trouble. Her temperature was between 100° and 101°, and it did not come down to the normal until the eighth day. She had a slight nocturnal elevation of temperature about three weeks after the operation, and expectorated a little purulent fluid, but there was no evidence of anything like empyema. Here was another case of acute gastric ulcer which was treated by laparotomy, and although peritonitis had commenced, you see that efficient drainage and irrigation resulted satisfactorily. By efficient drainage I mean drainage of the pelvis, drainage of each flank, and, if necessary, packing of the parts around the ulcer with gauze. By these means she was delivered from what undoubtedly would have been death if she had been left alone.

I will now read to you the notes of another case, which, I am glad to say, also ended in recovery. It was not an acute case, but a chronic one. This is a patient who originally was admitted under Dr. Rolleston's care, and by him was transferred to me She was suddenly seized with severe pain in the left side near the lower ribs while walking along the street. She was brought to the hospital in May, and was sick. On the previous Easter she had had an attack of pain, which was attributed to pleurisy. She continued to have pain in the lower part of the left chest, and had had a similar attack, which was also called pleurisy, two years before. That also was probably the pain of gastric ulcer. For the three years before admission she had suffered from anæmia and indigestion, the pain starting two hours after food, and getting worse until she took food again. The pain was especially relieved by hot drinks. Eighteen months before coming in she had had a severe abdominal pain and retching after eating. When she came in the abdomen was distended, moved slightly with respiration, and was rather tender in the epigastrium, but not all over. The muscles were resistent in the epigastrium, liver dulness

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slightly diminished. Dr. Rolleston got me to see her on the day following admission, with a view to operation. He thought, and quite rightly, that it was gastric ulcer which had perforated, and when she was under an anæsthetic I found distinct dulness in the left hypochondrium. On this account I cut down in that region rather than in the middle line. On opening the peritoneum there was a considerable quantity of pus and contents of the stomach in a subphrenic abscess. The abscess tracked upwards towards the posterior wall of the stomach, and apparently was localised. The perforation was not found. It was not found for two reasons, one was that it would have been difficult to find, and another that I did not try to find it. And I may say here that in all these cases of chronic perforation with a so-called subphrenic abscess, the surgeon should not try to find the ulcer if it is not apparent under his eye. He should not go searching for it because if he does he may generalise what is at the time of his operation a localised collection of pus and stomach contents, and he may infect the general peritoneal cavity. So here, when the general cavity had been shut off with gauze packing, a counter-opening was made behind in the loin, and two large drainage-tubes were inserted. This case, hen was a subphrenic abscess in connection with perforation of a gastric ulcer of fairly old standing, which I drained by a front incision over the dulness and by a posterior incision which I made in the flank. Next day her temperature went down to the normal; her pulse was 108. I removed the packing of gauze three or four days after the operation, and I may say, to make a long story short, she got well. She was anæmic and had irregular menstruation, and obviously had suffered trouble of the same nature eighteen months before her admission. On that occasion, very probably the stomach ulcer had leaked slightly, and this was the commencement of the subphrenic abscess. She never had any trouble in the lungs or pleura, although she spat up some purulent matter some twenty-four days after operation. I mention the lungs because I want to impress upon you the fact that people who have subphrenic abscess, and who die as a result, die from lung trouble rather than from any abdominal trouble. These subphrenic abscesses in connection with gastric ulcer hardly ever burst, but whether they are in the great sac of the peritoneum or in the lesser sac, they will travel up to the under surface of the diaphragm, perhaps the liver will be pressed down by them, and then from the abdomen the pleura becomes contaminated, and empyema results; and the patients will die, if neglected, from the empyema and the chest complications as much as from those in the abdomen. These subphrenic abscesses in connection with such a case as I have related are worth considering. I have had a diagram put up here which shows a vertical section of the peritoneum. You will see that in front of the stomach there is nothing but abdominal parietes; posteriorly, of course, there is the pancreas and duodenum and other structures. You seldom get adhesion between the stomach and anterior abdominal wall, though you do occasionally. I operated upon a case a little while ago where there were such adhesions, but sometimes when there has been a chronic lesion of the stomach you will get the great omentum adherent to the anterior abdominal wall. Supposing that the great omentum is adherent to the anterior abdominal wall, you have a cavity which is bounded above by the diaphragm and liver, and limited usually by the falciform or suspensory ligament of the liver on one side of it, in front by the abdominal parietes, and behind by the great omentum and the stomach. If you have a chronic perforation of the stomach in front you will have a collection of stomach contents, gas, and pus in this cavity. Let us suppose that the stomach ulceration is chronic, and that it occurs behind; then you will have a collection of pus in the lesser sac of the peritoneum, and this abscess may make its way downwards, separating the stomach and transverse colon, and may present at the lower part of the abdominal wall. When you have perforation in the anterior wall, the abscess will cause bulging with resonance, and a line of induration at its lower margin, where the great omentum is adherent to the anterior abdominal wall. It will present at the upper part of the abdomen. When perforation has been posterior, the abscess comes forward in the lesser sac, and will present lower down. Those two forms of subphrenic abscess may follow chronic perforation of stomach ulcers. For either of them to occur it is necessary that the foramen of Winslow be blocked up; if it is not, in anterior perforation the pus and stomach contents may pass

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into the lesser peritoneal sac as well as into the larger. If it is a posterior perforation some of the contents of the stomach may pass into the general peritoneal cavity as well as into the smaller sac. As a rule the foramen of Winslow is blocked in these cases of subphrenic abscess due to chronic perforations of the stomach.

Well, gentlemen, I have read you two cases of the acute variety which have been successfully treated by laparotomy, and one of the chronic variety, where also I have been lucky enough to have a successful result. I say lucky enough, but at the same time I believe that if these cases are treated early by laparotomy, and time is not wasted, nearly every surgeon ought to be "lucky" enough to have success. It is in the cases in regard to which time has been allowed to lapse before the surgeon has been called in, where general peritonitis is present, where the patient is collapsed and moribund, that you get deaths. I will read you the account of such a case which occurred in my own practice here. It was an acute case which came to the hospital practically moribund in 1901. On March 2nd of that year she came complaining of pain in the epigastrium, right iliac fossa, and about the umbilicus, followed by vomiting. The bowels were constipated. On the 3rd she was collapsed, the abdomen distended and tympanitic. When once the abdomen becomes tympanitic it means, as a rule, that peritonitis has started, especially when it is associated with rigidity of the abdominal wall. There was in this case free fluid in the peritoneal cavity; the abdomen was rigid, tender, and practically motionless on respiration. Liver dulness was normal. I opened the abdomen in the middle line below the umbilicus, and found acute general peritonitis present. The small intestine was for the most part distended, and the right iliac fossa was covered with recent lymph. The appendix seemed normal. I opened the abdomen below the umbilicus first of all to examine the appendix, as it was not certain what structure had perforated. The incision was prolonged upwards, and in the upper part of the abdomen quantities of turbid fluid and stomach contents were found, also a large ragged perforation on the posterior wall of the stomach, which was disclosed by an opening through the great omentum. In this case, then, I found perforation on the posterior aspect, and I had to go through the great omentum to get at it. When you find no perforation on the anterior wall you can examine the posterior wall by cutting through the great omentum, as I did in this case which I have related to you, and which I believe is the best course, or by going through the lesser omentum, or by passing your finger through the foramen of Winslow and examining the stomach through that aperture. I think that examination through the great omentum is the easiest. The walls of the stomach were very rotten. The parts were packed with gauze, Keith's drainage-tubes inserted, and the wound closed. The patient was very collapsed before the operation, and, indeed, practically moribund, and in spite of injections of strychnine and of saline fluid she died shortly after the operation. There had, then, been perforation and general septic peritonitis. This latter was well marked, and her case was practically hopeless before the operation. If she could have been operated upon a little earlier there is no doubt she would have had some chance of recovery, and, I think, a comparatively good chance. The case will serve to show you how these cases of acute perforation of gastric ulcer terminate when they are left too long without operation.

When gastric ulcer is of the acute variety there is a little punched-out opening on the stomach, such, for instance, as in this preparation. If, however, it is chronic, the ulcer may attain a very considerable size, as you see in this specimen. Some chronic ulcers of the stomach have been described as large as the palm of one's hand. When they are acute or chronic and perforate they give rise to trouble and symptoms similar to those I have described. When the ulcer is chronic it may cicatrise, and give rise to other trouble, such as stenosis about the pylorus, or it may lead to that extreme condition known as hour-glass contraction of the stomach. That, however, is not a condition about which I shall speak this afternoon, because I have no time for it, but I may say it is a condition which surgery steps in to try and alleviate. Here is a very typical chronic ulcer of the stomach. You see in this preparation when it is chronic it is not punched out so cleanly as when it is acute, and there is considerable thickening and infiltration about its edges.

The diagnosis of stomach ulcer I shall not say

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anything about; that, perhaps, is more for the physician to consider. We, as surgeons, have to deal with stomach ulcer not only after it has perforated, but physicians are now-a-days more and more calling the surgeon in to deal with cases before they perforate, so as to prevent the shock, collapse, and danger which results from perforation.

I should like to mention one other case in which I was called in to operate for a perforated stomach ulcer, and where I did operate but found no ulceration or perforation. Some of you may perhaps recollect that case. I was summoned to it at two o'clock in the morning in the medical wards. The patient had been there a fortnight with a history of hæmatemesis and melæna, epigastric pain, and all the symptoms of stomach ulcer. On the night in question she had a most violent attack of pain; she was lying in bed and groaning. Her distress was apparently very real, and it was therefore thought the stomach ulcer had perforated. I opened the abdomen, but found, after careful examination, no evidence at all of stomach ulceration. I at once stitched her up, and she was put back to bed. After the exploratory incision she appeared to be quite cured of all her previous symptoms. It was a case of neuromimesis. Probably she had had some ordinary indigestion. I examined the stomach very carefully, and there was no trace of any disease in the shape of ulceration. She was in the ward next to a woman who was suffering and really dying from cardic pain. This cardiac patient had been groaning with agony, and had been closely imitated by the girl with the supposed perforation of a gastric ulcer. I did think of the possibility of the latter's symptoms being neurotic, but the distress seemed to her physician to be too real for that. She was very satisfied with the result, and probably the exploration cured her of her imaginary troubles.

Among other conditions from which you may have to differentiate gastric ulcer are perforations of the appendix. But those, as a rule, involve another part of the abdomen. You are all more or less familiar with McBurney's point, and with the significance of pain about there, and rigidity of the right iliac fossa. At the same time, if you have perforation plus general peritonitis, it is difficult sometimes to say whether you are dealing with gastric ulcer or with perforation of the appendix. You should explore in either case. I had a case two years ago in which the stomach was affected, and where I confess I thought the appendix was involved; accordingly I opened the abdomen below the umbilicus. The symptoms previously had been such as usually accompany a perforated appendix, that is to say, abdominal pain and vomiting, the pain being chiefly referred to the right iliac fossa. There was also a rigid abdomen and apparently signs of commencing peritonitis, with intestinal obstruction. I opened the abdomen, but found nothing but a huge dilatation of the stomach, but no perforation of any kind. The stomach came right down to the iliac fossa and went up again in a V form. There was a considerable apparent œdema of the duodenum and the intestines were collapsed, otherwise nothing whatever was found. There was no intestinal obstruction, no perforation of the appendix or of the stomach, or anything else. It was one of those rare cases of acute dilatation of the stomach. I have not yet published that case, although I feel I must do so, because it is the only case of the kind we have had in this hospital. The patient was a woman æt. 27, and there was nothing of importance in her previous history, and she had never had any previous gastric trouble. The onset of her disease was quite sudden, and when she was admitted it was thought to be a case of fulminating appendicitis. There was profuse and continuous vomiting, which, of course, is different from the symptom of acute perforation of stomach ulcer. It was a dark greyish vomit, and evidently contained bile. It was not offensive. The bowels were collapsed, the pulse small, rapid, and feeble. She passed no urine at all while in the hospital, and had all the physical signs described by Dr. Fagge as connected with acute dilatation of the stomach. She was collapsed, restless, and conscious, and suffered but little pain. The pulse was 135, and it suggested peritonitis. The abdomen, however, though distended and tender, was soft to palpation. There was dulness and apparent fluctuation above the pubes and in the right iliac fossa. The epigastrium and right hypochondrium were full, and the amount of distension varied with the vomiting. There was a history of three days' duration. The physical signs and general appearance of the patient suggested peritonitis. I operated, and the incision was made over the

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appendix where the fluctuation and dulness were apparently present. On opening the abdomen an enormously dilated stomach presented. The cardiac end of the stomach was held up, and the rest formed a V-shaped curve which completely filled the abdomen; it contained gas in large quantity and fluid. At the operation the stomach was emptied by means of a tube passed by the mouth, but the patient died about four hours afterwards. Examination of the abdominal viscera post mortem showed the stomach to be low down, elongated, and slightly distended, much smaller than at the operation. All the intestines were collapsed and retracted. On opening the stomach it was found to contain mucus and a greenish fluid. The mucous membrane was congested, especially over the greater curvature near the pylorus; the rest of the bowels showed congested and œdematous patches of mucous membrane. The duodenum was much thickened and œdematous, the rest of the organs were healthy. That was a very extraordinary case of acute dilatation of the stomach. The number of such cases which have been recorded is very limited. Mr. Tucker, my late house surgeon, to whom I am indebted for these notes, has collected the records of twenty-six such cases, all of which were fatal, and that in a very rapid time. I only mention that to you now as illustrating one of the acute conditions of stomach which you have to bear in mind, and which you might possibly have to deal with.

Remember that acute perforation of a stomach ulcer is a condition which any one of you may suddenly have brought before you, perhaps in country practice, at any time in after life; and the early recognition and treatment of such cases is so all-important that I think every one of you should so educate yourselves as to be able, if necessary, to operate at once; and if you operate on the lines I have laid down this afternoon, operating very early, *i. e.* at once, seeing that you have free drainage, not only of the immediate neighbourhood of the ulcer but also of the flanks and Douglas's pouch, packing the parts if necessary with gauze; if you cannot suture the rent in the stomach there is no reason why any one of you should not have successful results. I do not think this kind of case should wait for eight or ten hours until a hospital surgeon can be procured to do the operation. This operation should be performed by the practitioner of the future just as now tracheotomy is, for the symptoms may be equally urgent, and the chances of a catastrophe happening from delay are quite as great. If, as I pointed out, the operation be performed within a few hours of the onset of the acute symptoms, the prospects are good, as shown by the cases I have narrated to you. But if operative procedures are not attempted until the patient is moribund and practically *in extremis* from the general peritonitis which has supervened, nothing can do any good.

Note, April 8th, 1902.—The case first mentioned completely recovered. Her convalescence was slightly retarded by some swelling and œdema of the left leg.

What Constitutes Septic Poisoning in Accident Policies.—Kemper, G. W. H., 'Philadelphia Medical Journal,' vol. ix, No. 7, p. 328, February 15th, 1902.—The writer reports his experience in making a claim against a company which had insured him against septic poisoning. He was attending a surgical operation and accidentally infected his ear, which was the seat of an eczematous eruption. When the claim was presented a polite letter was returned stating that the policy did not cover diseases of any kind. This led to an investigation of the clauses in accident policies which are issued to physicians covering septic poisoning. One company issues policies, but restricts the septic poisoning to such cases as do not result from the physician's own carelessness. Another regards the indemnity as payable only when the septic poison has been received as the result of an infection of a wound received in an operation. A study of the adjudicated claims and of the terms of the various policies leads the writer to the conclusion that the term "septic poisoning" in an accident policy is simply put in for the purpose of securing policy holders, but with no idea of paying any claims which may arise under this clause. In most cases it is so artfully worded that there is no difficulty in the insurance company avoiding payment of the claim.—*Medicine*, April 1902.

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A CLINICAL LECTURE

ON

THE TREATMENT OF NÆVI BY ELECTRICITY.

Delivered at St. Bartholomew's Hospital, November 18th, 1901.

By H. LEWIS JONES, M.D., F.R.C.P.,

Physician in Charge of the Electrical Department of the Hospital.

GENTLEMEN,—Although nævus is not a thing which is ordinarily dangerous to life, yet in the course of your work you will find that its treatment is sometimes extremely difficult. Nævus commonly occurs in positions where it cannot be dealt with satisfactorily except by electrical means, and in this lecture I propose to consider the details of the methods in which one may treat a nævus by electricity. You must not think that all nævi need to be treated by means of electricity, because there are a great many which can be dealt with more expeditiously in other ways. If excision is feasible, if the nævus can be completely removed by the knife, that is the best method, because there the whole of the affected part can be got rid of at one operation. When that is impossible one must be content to try to arrest the nævoid growth without producing too much in the way of scarring, for it often happens that the treatment of nævus involves not only the destruction of the nævus, but the doing it in such a way that no obtrusive scar shall be left behind. To begin with, nævus is twice as common in female children as it is in males. Again, nævus is much more common on the head and face than on any other part of the body, and so you will understand that it may be very important from your point of view, and from the parents' point of view, that you should not disfigure a child's face if you can possibly avoid it. When a nævus is round and button-like and small, it may be excised. When it is diffused, and occupies a part such as the eyelid or the nose or the lip, as it so often does, there is no tissue to spare, excision is out of the question, and the nævus has to be destroyed in some other way. Another thing which makes the treatment of nævus very troublesome is the tendency of a nævus in a young baby to grow. You may operate and destroy a nævus completely, so far as you can see, and yet, after three or four weeks, when your wound is healed, you may find fresh spots of nævus springing up all round the margin of its original position, outside the area occupied by the original growth. No doubt those portions were there in an embryonic form at the time of the first operation, but were invisible at that time. These recurrences and new outgrowths are an annoying feature of the treatment of nævus.

In treatment by electrical methods, we may either use the electricity directly, by passing the actual current into the tissues, and so destroying the nævus by electrolysis, or indirectly by employing the electricity to produce heat in a galvano-cautery, with which to cauterise the nævus.

For anyone who is but slightly experienced in electrical matters it is best to use the cautery and not electrolysis for many of the nævi which you will have to deal with. Many cases can be treated very nicely with the galvano-cautery, if you know how to do it. Here I show you a battery suitable for heating the cautery. I do not propose to describe the battery to you now, but it is an accumulator of the regular type for cautery work. Here is the platinum point, which is made hot when the current is turned on, and the nævus is treated with this hot point. You will notice that my cautery and holder have the form of a small light apparatus. This other one on the table, which is the common form in general use, is very stiff and clumsy to use for work on small nævi. Mine, you see, is of the shape and size of a penholder. Care must be taken to get the point to the proper temperature, and that is a little practical point of interest. It should be at a dull red heat; if it is white hot it will cut the tissues and they will bleed, and this bleeding will be troublesome and will obscure your view. That is not the case if the point be heated to a dull red. If it is not hot enough the tissues stick to the point, but a point which is at the right heat in the air will be much cooled when thrust into the tissues. Therefore make the point a little hotter than you require it, and cool it down by blowing upon it while in the act of putting it into the nævus. The cautery is said to be an excellent way of arresting hæmorrhage, but my experience is that if your nævus begins to bleed during the operation you will find it very difficult to arrest, even with a cautery.

The next point is with regard to the method of

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using the cautery. The way one is tempted to attack the nævus is by searing the top of it by rubbing the cautery over it. By rubbing the cautery over the top of the nævus you will give it a brown discoloration, and it will look as if the tissue had been destroyed; but if you stop at that point you will find that in a few days the burnt crust will come away and there will be remaining a nævus apparently unaltered, and the process all has to be done over again. A nævus has a definite thickness, and to simply burn the top of it is not enough. The heat produces an intense constriction of all the blood-vessels, which makes the nævus seem as if it were destroyed, but you will find that it is not so when the scar comes away. The same remark applies to caustics. I often see nævi in the electrical department of this hospital which have been previously treated elsewhere outside with nitric acid, which has left a scar, but the nævus has not been destroyed. It is necessary to push the cautery through into the depth of the nævus; see that it goes in as deeply as the nævoid tissues extend.

Next comes the method of treatment by electrolysis, and then we will touch on the relative advantages of the two methods, and consider what kinds of nævi are suitable for the one and for the other respectively.

When you electrolyse a nævus you destroy it not by heat but by chemical action. The current in flowing between the needles which are inserted into the tissues liberates caustic chemicals, which gather round the needles and destroy the nævus. The amount of chemical substance liberated is proportional to the amount of current which passes through the nævus and to the length of time during which it is allowed to pass. The substances liberated at the positive pole include hydrochloric acid, phosphoric acid, and sulphuric acid, while at the negative pole will be liberated potash and soda, and so forth. It is those chemicals which do the work, and not the flow of current from one pole to the other. If you were to examine an electrolysed nævus microscopically, you would find the destruction had taken place around the needles, the intervening parts being unaffected. I will demonstrate that to you by means of litmus paper. Here is a piece of wet litmus on a glass plate, and on it I lay my needles for a few seconds. You see three blue streaks where the three negative needles have touched the paper, and two red streaks where the two positive needles have been; but between the marks there is no change. In the nævus you may use either both poles or only the positive or only the negative. If you use only one pole, then the circuit has to be completed through the patient by means of a pad wetted with water. You put the pad on the patient's back, and you attach to the other pole a platinum needle or needles like those I have here.

There are certain points to be considered in the choice of a method of treatment. If you use both poles you have the advantage that your current is confined to the region of the nævus. It only flows from needle to needle; there is no general passage of current through the rest of the body, and that is sometimes an advantage, because with a patient under chloroform there may be a certain amount of shock when using the pad, especially if the nævus be on the head or the face. If your pad were on the back, and your needle on the cheek or nose, a considerable current would pass in the child's neck between the pad and the needle. By the use of both poles in the nævus you avoid this risk of shock. But it may not be always convenient to introduce both poles into a small nævus. If the nævus is small you do not require a strong current, and you want to do fine work with one needle, and in this case you can use a single pole and a pad. With larger nævi it is better to use both poles in the nævus. As it requires a good deal of dexterity to introduce a number of separate needles into a nævus, and to manipulate them during treatment, I had made for me a needle-holder, which I show you here. It is a fork-like arrangement, and four or five needles can be screwed into the holder so as to be alternately positive and negative. By this means one can handle the needles easily, and it is very useful in the treatment of large nævi. One can vary the number of needles to suit the size of the nævus, and the current is limited to the nævus, and has no effect in producing shock. The difference in action between the positive and negative poles, although very appreciable, is not important; both poles destroy, the positive by the acids it liberates, and the negative by the alkalies it liberates. The positive pole, which liberates the acids, coagulates the tissue round about it, and the positive needle consequently adheres somewhat to the tissues, and

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in withdrawing the needle afterwards you may get bleeding. Very seldom is there any bleeding from the negative pole, and on this account when using one pole only for fine work it should be the negative one.

The battery for electrolysis must be a constant current battery. That is a very elementary fact, but you must pardon my mentioning it because I am continually being asked whether an induction coil will do for the electrolytic treatment of nævi. The induction coil will not do, because the current which it gives out produces no electrolysis, and, therefore, no destruction. This portable battery which I show you provides everything which is necessary for the treatment of nævi. It contains thirty small Leclanche cells. For doing electrolysis you will seldom need more than twelve or fifteen cells.

With regard to the magnitude of the current, if you watch the galvanometer of your battery during the electrolysis, you will find it requires from twenty to thirty milliampères. You will also find that the current which a certain number of cells will drive through the nævus will depend on the number of needles used, and the depth to which they are introduced. Therefore the more needles you use, and the deeper you introduce them, the larger is the current which you will be using. Always regulate the strength of your current proportionately to the extent of your needles. It is the density of current or strength per unit of needle surface which determines the amount of the destructive action, and a simple rule to lay down is that the current should be about thirty milliampères per inch of positive or negative needle. That will give you an average strength of current to work by.

How are you to know when the treatment has been carried on enough? If the needles are put into a nævus and the current is allowed to flow for a long time the whole nævus will be so destroyed as to slough, and that is not the ideal result. Your aim should be to produce plastic inflammation in the nævus and to keep short of complete destruction. If you make the whole nævus slough you make an ugly scar, and your result will therefore be second-rate. If you do it properly you will have a firm but inflamed area, obliterating the nævus, but causing very little scarring. To learn how to electrolyse a nævus nicely there is nothing like practice; if you watch the process as it is going on you will see a little greyish zone forming round the points of entry of the needles; it gets bigger the longer the current is continued. If you change the position of the needles when this zone is about one eighth of an inch across you will get destruction with little scarring at that needle hole, but if you wait until it is a quarter of an inch across you will have sloughing there, and there will be a scar left afterwards. So if the nævus is large enough to require it, change the position of your needles during the electrolysis and introduce them afresh, until you have completely covered the ground. Remember it is the edge of a nævus which grows most rapidly, and it is useful to insert your needles all round the margin, and a little outside the edge rather than inside it, so as to attack the growing edge thoroughly. An advantage of the fork needle-holder is, that with it the needles stand parallel to each other, and so the current is evenly distributed. If two needles be put into the tissues in a converging direction, the greatest amount of current will pass at the point where the needles are closest to each other, and the electrolysis will be greatest there; whereas if they are parallel to one another, the current and the destruction of tissue will be equal along the whole line.

Now a few words with regard to the relative advantages of the constant current and galvano-cautery. I have been treating nævi at the hospital for the past eleven years, doing several nearly every week. At first they were nearly all done by electrolysis, but now I am doing nearly all of them by galvano-cautery. The change from the one method to the other is instructive when one examines it. The treatment by electrolysis is not quite so rapid as that by the galvano-cautery. Electrolysis is rather more likely to be followed by recurrences of the growth than when the treatment is by the cautery. The destruction by electrolysis is less than by the cautery. Those are the two essential points of difference between the two methods. If you have a rapidly growing nævus in a part of the body which is not important from a cosmetic point of view, such as on the legs or the trunk where the clothes will cover it, and where a little scar is not particularly important, attack it vigorously with the galvano-cautery and destroy it as thoroughly and as quickly as possible. If you have to treat a nævus on the eyelid, or on the nose

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or the lip, where there is very little tissue to spare, and especially if it be a small nævus and is not growing very quickly, choose electrolysis, because you will destroy less tissue and produce less scarring. But you must tell the friends that the treatment may be a little slower, and you must prepare for possible recurrences. So, again, there are nævi which are almost entirely subcutaneous, and are covered by sound skin. In such a case it is desirable not to destroy the skin more than can be avoided; therefore you will choose electrolysis rather than the cautery. On the other hand, you will have nævi where the skin is nævoid and vivid red in colour. This must be destroyed, and for such a case you should use the cautery.

I may mention one or two special varieties of nævus. First with regard to nævi in the middle line of the body. These are not at all uncommon in the middle line of the head and of the face, especially at the root of the nose, in the upper lip, and in the lower lip, and again they are found lower down about the genitals. These middle line nævi usually grow very rapidly because they get a blood-supply from both sides of the body, and therefore they require more severe treatment than nævi which are not in the middle line. You will also occasionally be confronted with nævi of the fontanelles. These require careful treatment for obvious reasons. The cautery or the needles must be pushed in horizontally and not vertically. Otherwise there is no special danger about them.

A third point I wanted to speak about are nævi in adults. From time to time you see an adult who has a nævoid tract of tissue in his body. Often these cases are very extensive, thus the entire arm or leg or the side of the face may be nævoid. Be very careful about these things. Do not be too ready to attack them at all; it is better to let them alone if they are not growing, and these nævi often remain quiescent in adults. These nævi in the limbs of grown-up people are sometimes composed not of capillaries but of large irregular venous channels, and may give troublesome hæmorrhage. A nævus in the orbit is also very difficult to treat satisfactorily. Among curiosities I may mention one of a nævus of the iris. I have only had to deal with one, and fortunately that did not grow, and so we were able to leave it alone. The treatment of such a case would be excessively difficult.

Now with regard to the question as to when to operate on a nævus. When a baby is born with a nævus, the advice often is given to wait and see if it grows. Treatment is postponed and postponed, and in some cases a nævus which was very small at birth is perhaps allowed to grow until it attains the size of a shilling or a half crown, and by that time it has become an extremely formidable thing. This is the wrong way of dealing with nævi. If you are going to treat the nævi of infants do so at once. A large nævus on such a part as the face is far more difficult and troublesome to deal with than a small one is, and it may take six months of time and several operations to get rid of one which has been neglected and allowed to grow big. When you are convinced that a nævus is growing, the sooner it is removed the better, and be sure you treat it thoroughly. The first attack upon the nævus must be your chief opportunity. At the first operation you should succeed in destroying at least half or two thirds of the growth if not the whole of it. I invite you to come and see nævi treated in the electrical department on Thursdays. After operation they are painted with collodion and iodoform, and on the following Tuesdays they receive their first dressing, and then is the time to see the effect of the operation, and to estimate the probable success of your treatment.

Empyema.—Dr. Samuel Lloyd states that 58 per cent. of the cases of empyema in the babies' wards of the New York Post-Graduate Medical School and Hospital had shown the pneumococcus. He thought nearly all surgeons would agree with the treatment laid down by Dr. Dowd, but he would differ a little as to the technique. The anæsthetic should be stopped on exposing the pleura, and the finger used to break up the adhesions. The lung then expands, and the child, coming out of the anæsthetic, cries or coughs, and so assists in this expansion of the affected lung. It was remarkable how promptly and well the lung would expand under this simple breaking up of the more recent adhesions. In the cases of long standing the adhesions were so firm that nothing short of an Estlander operation, or similar radical measure, would suffice.—*Medical Record*, April 5th, 1902.

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CLINICAL OBSERVATIONS ON SOME CASES OF EARLY TUBAL PREGNANCY.*

By FREDERICK J. McCANN, M.D.Edin., F.R.C.S.Eng., M.R.C.P.Lond.

Physician to In-patients at the Samaritan Free Hospital for Women, London.

MR. PRESIDENT AND GENTLEMEN,—I have chosen for my remarks to-night a subject of common interest and far-reaching importance to those engaged in the busy practise of their profession. For it is to the better understanding of the early signs and symptoms of tubal pregnancy that we look for guidance in our methods of treatment.

It is most important that a more complete record of early cases should be at hand, in order that accurate diagnosis may anticipate some of those terrible complications associated with rupture or leakage of a gestation sac. To this end the present short series of cases may prove to be not without advantage to those who may not have opportunities of acquiring special knowledge in this department of practice.

CASE 1.—A lady æt. 27 years was sent to me by Dr. Laing-Gordon on November 16th, 1897. She had been married three years, and was sterile. Three weeks before I saw her she had a sudden attack of pain in the left iliac region, accompanied by sickness and faintness. At that time her menstrual period had been delayed for fourteen days. After this attack she was confined to bed and attended by a medical man in the country, who thought she had "inflammation of the bowels." On bimanual examination the uterus was found to be well-developed, and situated forwards and to the right. The right ovary was large and prolapsed, and the right tube thickened. In the left posterior quarter of the pelvis there was an elongated elastic swelling. As the history pointed to a leakage of the contents of this swelling into the peritoneal cavity, operation was advised.

On November 20th the abdomen was opened—removal of tube, ovary, and clot,—on left side. Right tube thickened. Excellent recovery.

In June of last year (1901) patient gave birth to a full-time male child, and had an easy labour.

* Delivered before the Sydenham District Medical Society.

CASE 2.—Married woman, æt. 37 years. Had had four children, the youngest being two years old. Since the birth of her last child her menstrual periods had been regular until they ceased for three months.

On November 13th, 1897, she felt ill, complaining of acute continuous pain in the right inguinal region and thigh. The thigh was also swollen. On November 17th she had a slight vaginal discharge of bright red blood. The swelling in the thigh disappeared. The pain continued to increase, being referred to different parts of the abdomen, but chiefly the right iliac and umbilical regions. The red vaginal discharge ceased until November 21st, when a greenish discharge was observed. The temperature varied from 99° F. to 101° F. Pulse 100 to 120. The bowels were constipated. Micturition difficult and painful.

I saw the patient on December 6th, 1897. She then complained of intense pain in the right side of the abdomen. She was very blanched. Her skin was cold and clammy. The temperature was 101° F. The abdomen was slightly distended and very tender on palpation. There was dulness on percussion, extending upwards from the right iliac region to the level of the umbilicus. Bimanual examination was very difficult owing to pain.

Examination under anæsthesia before operation revealed an elastic elongated swelling, occupying the right posterior quarter of the pelvis; and a rounded boggy swelling, which appeared to move with the uterus, was also detected. This swelling extended above the pelvic brim on the right side. Operation: abdominal section, removal of dilated right Fallopian tube filled with blood-clot, enclosing a central cavity. A large mass of blood-clot was found at the expanded ampullary end of the tube. About a pint of blood-clot was also removed from the abdomen. The patient recovered, and has remained well. Sections from the tubal wall and contained clot showed chorionic villi.

CASE 3.—Mrs. A—, æt. 30 years, was sent into the Samaritan Hospital by Dr. Gore Reynolds on January 18th, 1901. She was a stout woman, who looked very ill and bloodless on admission. She had been married four years, and had a child a year after her marriage. Her health had been good until September, 1900, when she had a

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"miscarriage," being then, as she said, three months pregnant. Eight weeks before her admission she was attacked with severe abdominal pains. At this time she was supposed to be six weeks pregnant, and fourteen days later it was alleged that she had another miscarriage. Since this attack she had been losing blood daily from the vagina. The discharge, on examination, was noted to be of a dark colour. The pain, which increased at intervals, also persisted. On examination *per hypogastrium* a swelling was felt, extending upwards on the right side to a point midway between the level of the umbilicus and the symphysis pubis. Bimanually this swelling was found to be continuous with another swelling found in Douglas's pouch. The uterus was enlarged, and displaced forwards and to the left. The cervix was softened.

The history of the two miscarriages was indefinite, as no description of ovuline structures being passed was obtainable.

From the symptoms and physical signs a diagnosis of ectopic pregnancy was made, and operation advised. As the swelling presented in Douglas's pouch, I decided to incise the posterior wall of the vagina and explore.

The swelling was found to be a large hæmatocele encapsuled. It was emptied of blood and clots, and the edges of the capsule fixed by chromic catgut sutures to the vaginal wall. The cavity was packed with gauze. The gauze was removed four days later. The patient recovered, and has remained well.

Previous to the operation the patient was kept in bed for a few days and carefully watched. The swelling was observed to increase in size, therefore operation was advised.

CASE 4.—An anæmic-looking woman, æt. 35 years, was admitted into the Samaritan Hospital under my care in October of last year. She had had one child born four years ago, but no miscarriages. On August 8th, having missed her period for six weeks, she had severe bleeding from the vagina, blood and clots being passed. A piece of flesh described as being the size of the palm of her hand, was also passed. Previous to this attack she had noticed that her breasts were painful, and she had morning vomiting. During the attack of hæmorrhage she suffered considerable pain, which persisted for some days. The pain was situated in the left iliac region, and was so severe that it caused her to faint on three occasions. The pain existed at intervals until her admission. A discharge of *bright blood* continued to issue from the vagina, and existed when she was first seen by me. The breasts were stated to have become smaller, and the pain in them had subsided. A lump had been detected to the left side of the uterus on September 12th, and an enlargement of the womb was also noted. This lump was observed to have become smaller on October 6th. On examination an elongated movable swelling was palpable on the left side of the uterus, occupying the left posterior quarter of the pelvis. The uterus was slightly enlarged.

The patient was kept in bed under observation for a few days. During this time the discharge of blood continued, the pain in the left iliac region persisted, and the swelling was noted to have increased in size.

A diagnosis of tubal mole was made, and operation advised.

The posterior vaginal *cul de sac* was incised, and the peritoneum opened. The fingers were inserted into Douglas's pouch, and the swelling drawn downwards. It was punctured with a trocar, and some dark blood evacuated. The left ovary, which was cystic, was also punctured. The tube and ovary were then pulled into the vagina, a pair of large Spencer Wells's forceps placed on the pedicle, and the tumour separated with scissors. A hydrosalpinx was found on the right side; it was punctured, ligatured, and removed. The forceps were left *in situ*, and a piece of gauze inserted as a drain completed the operation. Two chromic catgut sutures were inserted into the posterior vaginal wall to control hæmorrhage. The whole proceeding only lasted fifteen minutes. The gauze and forceps were removed in twenty-four hours. The patient recovered, and remains well.

CASE 5.—Mrs. J—, a fairly healthy-looking woman æt. 29 years, was brought to me by Dr. Platt on January 15th, 1901. She had been married four years. Previous to her marriage she suffered severely from dysmenorrhœa, which was not altered by marriage. A year later she stated that she had had amenorrhœa for two months, followed by blood-loss for three weeks, with constant pain.

Before I saw her she had missed her period for one week, and had severe pain in the left iliac

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region, accompanied by bearing down. Since then the bowels had been more constipated. She had for some time complained of dyspareunia. On examination an elongated movable swelling was palpable on the left side of the uterus. The uterus was not much enlarged. There were no breast signs and no vaginal discharge.

I regarded the case as one of tubal pregnancy, and accordingly recommended operation.

Operation January 23rd, 1901. Median incision. A rounded blood-clot was removed from the abdomen, and the left tube found to be dilated, and on the point of bursting close up to the uterus. On getting it up through the incision it was found to be very friable. It was ligatured and removed, with the corresponding ovary, but owing to the friable tissue extending to the uterine cornu it was necessary to insert a mattress suture of strong silk in this position to control bleeding. The peritoneal edges were then brought together with fine silk sutures. The appendages on the right side were not diseased. The abdomen was flushed with hot salt solution, and the wound closed. The convalescence was interrupted by a sharp attack of bronchitis, from which, however, the patient completely recovered, and left the nursing home well. [She had had bronchitis every winter for some years.]

The specimen removed showed an early tubal gestation situated at the uterine end of the left Fallopian tube. Sections from the tubal wall showed chorionic villi.

On December 3rd, 1901, at 12.30 a.m., I had an urgent message to go to Forest Hill to see this patient, and from the account the husband gave me I concluded she had a ruptured tubal fœtation. This opinion was shared by Dr. Parnell and Dr. Grayling, who had seen the patient before I arrived.

I found her extremely blanched, her radial pulse just perceptible, dyspnœa well marked. She vomited once in my presence. Her abdomen was slightly distended, and very tender on palpation. There was dulness on light percussion in the hypogastric and iliac regions. On bimanual examination (which was conducted with great gentleness) the pouch of Douglas was filled with something which gave the sensation to the finger as if pressure was being made on a feather pillow. The examination caused the patient to feel very faint. I accordingly concluded that the abdomen was filled with blood, and that an operation should be done as soon as possible.

As Dr. Parnell told me her condition was improving, we thought it well to make careful preparation for an operation.* On opening the abdomen dark blood and clots welled up out of the wound. I at once introduced my hand, grasped the right tube, brought it out of the wound, and clamped it with an elbowed Spencer Wells's clamp. The abdomen was then flushed with several pints of hot salt solution in order to clear out the blood and clots. The pedicle was ligatured after removal of the clamp, and the wound closed.

The history obtained from the patient was that while at stool she was attacked with violent abdominal pain and faintness. She had missed her period for one week, followed by discharge of blood for three weeks, before this attack. Since her previous operation she had enjoyed excellent health.

The patient rallied well after the operation until the third day, when she complained of severe stabbing pain in the right side of the chest. There was dulness on percussion in this situation, and loud friction could be heard.

Signs of pneumonia soon developed, and she died on the fifth day. At no time had she any abdominal signs, and there was entire absence of sickness.

The gestation was situated in the same position in the tube as on the left side, namely, close to the uterus.

1. The diagnosis of tubal pregnancy before there is hæmorrhage into or around the ovum may be made by attention to the following signs and symptoms:—A patient not beyond the child-bearing period, and one in whom pregnancy is possible. There will be a history of amenorrhœa, changes in the breast associated with early pregnancy, together with a slightly enlarged uterus and a swelling on one or other side of that organ in the position of the corresponding Fallopian tube. The uterus can be proved to be empty by the passage of a sound, but I would not advise this to be done. Sterility, absolute or relative, is not uncommon, and some years may have elapsed since pregnancy has occurred.

* The anæsthetic was administered by Dr. Grayling.

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2. Hæmorrhage into and around the ovum, forming what is now termed a tubal mole, is proved to be one of the commonest terminations of early tubal pregnancy. If the abdominal end of the tube remains patent blood can escape into the abdominal cavity. If the abdominal end be closed the tube will become distended and burst. This bursting, however, is more probably due to the growth of chorionic villi into the tubal wall, with consequent weakening of the wall in that situation. When this occurs in addition to the signs of early pregnancy there is pain in one or other iliac region, and hæmorrhage from the vagina. The character of the hæmorrhage is most important. It is occasionally profuse and red, and may be mistaken for a uterine abortion. Here a careful bimanual examination will exclude any periuterine swelling. The hæmorrhage, however, is generally dark in colour, moderate in amount, and steady in its rate of flow. Gushes of bright blood may exceptionally occur. Further, a history of the passage of some membrane, either as a complete uterine cast or in two pieces or in shreds, may be obtained. The tubal swelling enlarges markedly by recurrent hæmorrhages and by the formation of a hæmatocele. It may later become smaller, as the clot contracts only to enlarge again with the advent of fresh bleeding.

Fresh hæmorrhage causes sudden pain and transient peritonitis.

If the extravasation of blood be considerable the uterus is displaced backwards, forwards, or to the side, according to the situation of the extravasated blood.

Anæmia is caused by these attacks, and if oft repeated the pallor is most profound. Rupture of the tube, with severe intra-peritoneal bleeding, or complete emptying of its contents into the abdominal cavity through the dilated ostium may cause sudden death, or the patient becomes moribund unless relieved by operation.

Three important lessons may be learned from the cases described in this paper:

1. The importance of a careful bimanual examination in cases of threatened miscarriage.

2. The diagnostic value of a dark-coloured vaginal discharge.

3. The diagnostic value of the condition of the pulse in intra-peritoneal hæmorrhage from extra-uterine pregnancy.

As the first two have been already mentioned, a word must be said in conclusion concerning the value of the condition of the pulse as a diagnostic sign.

In severe intra-peritoneal hæmorrhage from extra-uterine pregnancy the radial pulse may be imperceptible. If it be perceptible it is of a character which betokens severe blood-loss. Moreover, the aspect of the patient is one of intense pallor. In cases of ruptured viscera or perforating ulcer the radial pulse is generally perceptible, small, and thready, indicating acute peritonitis. The patient's face is somewhat livid. The character of the pulse in ruptured tubal pregnancy or in tubal abortion approaches more to the type met with in cases of peritonitis when active bleeding has ceased, for the extravasated blood causes localised peritonitis.

Somnolence caused by an Ear Lesion.—Dr. Harland reports the case of a boy æt. 13 years, who would go to sleep, without feeling tired or out of sorts, at any time when his attention was not aroused by action. He would fall asleep in a store even while his order was being filled. The condition did not simulate *petit mal.* There were no vertigo, tinnitus, nausea, or headache. There was a small plug of cerumen in the left ear. Upon syringing the ear the wax came out readily, and a few drops of fœtid pus were found behind it. The drum was macerated, and had a large perforation in the lower posterior quadrant. Within a day, without other treatment, the unnatural sleepiness disappeared. A return of impacted cerumen was accompanied by some return of the sleepiness, according to the mother, though this was denied by the patient. The symptom disappeared after cleaning out the ear. The author ascribes the symptom to either meningeal irritation from the absorption of pus or to pressure on the labyrinthine fluid.—*Philadelphia Med. Journ.; New York Med. Journ.*, April 5th, 1902.

BURROUGHS, WELLCOME AND CO. have now issued a "tabloid" containining 3 grains of ammonium chloride and 2 grains of extract of liquorice. It is a good way of giving ammonium chloride because the unpleasant taste of the drug is disguised.

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WITH

MR. MARMADUKE SHEILD

IN THE WARDS OF

ST. GEORGE'S HOSPITAL.

MARCH 11th, 1902.

GENTLEMEN,—To-day, instead of a set clinical lecture I propose to make a series of practical remarks, which are suggested by certain cases which are, or have lately been in this hospital, and these I hope will be as useful to you as a more formal lecture on any one case of disease or injury. The first instance I draw your attention to is that of a man in Hunter Ward, who has a sinus in the chest wall, the result of operation for an old empyema. Unfortunately he is doing very badly, and to-day, as is not uncommon with patients who are progressing unfavourably, he blames the physician and surgeon for the empyema and all the bad results that have followed from it. I daresay some of you will be very surprised that I should have picked out such an apparently uninteresting case to speak about, because very little attention is shown by students to such "uninteresting" conditions as old sinus in the chest wall. A man in the surgery who has a sinus from an old empyema, the result of operation here or elsewhere, will hardly get a fleeting glance from any of you, but if he was fortunate or unfortunate enough to have a curious, rare tumour as big as a man's head in Scarpa's triangle he would certainly have a crowd round him. This is the direct result of our teaching being directed towards the examination halls, rather than towards what is of utility in practice. But let me tell you that an old sinus in the chest wall is of very grave import, because it means that operation has been performed for empyema, and that this operation has not been curative. Such a condition, indeed, is of far greater importance to you, as future practitioners, than any rare forms of disease. If you trace to the

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end the patients who suffer from these sinuses, you will find that a large number die, either of exhaustion, of hectic fever, or of bedsores, or they succumb to lardaceous disease of viscera, or to general tuberculosis. They are exceedingly serious cases, and bad ultimate results too often supervene after operations for empyema in adult patients, especially in the hospital class. Accordingly, I thought I would commence by saying a few words about empyema and its treatment. The first thing I have to impress upon you regarding empyema in adult patients is the extreme seriousness of the disease. I do not think there is any acute malady in a grown-up person much more serious than a collection of pus in the chest, and as you will certainly have this kind of case to deal with in the future, recollect that you can safely and truthfully give a guarded prognosis in empyema in an adult. You must reflect that it is a very serious condition, that it will require a long period of treatment, that several operations may be needful, and even with the best treatment a sinus may persist, and fail to heal.

The most common causes of empyema are as follows:—The disease is essentially a septic one in its origin, and consequently it is not uncommonly the sequel of certain fevers, as smallpox, scarlet fever, or typhoid. I suppose in such cases the septic organisms gain access to the circulation through the lung, or through the tonsils or bronchi, and may thus infect the pleural cavity with a septic infusion. But yet it is true that very often in bad empyema cases the cause is unknown. A man will fall into bad health, and on examination of his chest he will be found to have a collection of purulent fluid, and you cannot tell why. I have no doubt that the true explanation of some of such cases is that they are associated with septic emanations from drains. I do not feel any doubt that many an inexplicable case of this kind is due to exposure to sewer gas. I have a theory—it may be wrong or it may be right—that some empyemata are created by a little carelessness on the part of ourselves; I mean that there is a grave risk of infecting a simple serous effusion within the chest by the introduction of septic needles. I have often observed in past days that a patient's chest had been tapped in order to draw off a serous effusion, and that shortly afterwards the fluid had re-accumulated, and the temperature became irregular. A needle was again introduced; this time the fluid was purulent. I fear in such cases a septic needle has been introduced, and has thus produced a purulent infection of the fluid in the chest cavity. Another point of importance is as follows:—The skin should be most thoroughly cleansed, and it should be incised at the point of puncture by a tiny sharp knife. A needle should never be thrust into the chest without incising the skin. This sounds a small matter; it is not so. If this is not done you carry into the pleural cavity in the aspirator needle a quantity of epithelial *débris*, and, likely enough, septic organisms. I need hardly impress upon you the importance of thoroughly boiling the needle. I am sure that the loose methods often employed in "tapping chests" are potent causes of serous effusions becoming purulent. There are other rarer causes of empyema, such as caries of ribs or spine, or the ulceration through the bronchi of foreign bodies, or the purulent effusion may be found in the course of a general pyæmia. But in a large number of cases, especially in adults, you will find empyema in association with pneumonia, and you will be at a lossto know the certain and actual cause of the mischief.

Now I will make a statement which is of the utmost importance to everybody engaged in the practice of medicine or surgery. Empyema is a commonly overlooked cause of wasting and prolonged fever of the hectic type. You will find hereafter in your practice that you will have a patient who for many weeks and months has a temperature which goes up at night, with sweating, and down in the morning, and you will be greatly puzzled as to the cause. Now in such a case remember to examine the chest with scrupulous care, and suspect empyema. This is at least of as much importance as counting the blood-corpuscles, or performing hazy experiments with the blood-serum. Other causes of such obscure febrile symptoms are undiscovered tuberculosis, pus in the gall-bladder, kidney, or prostate gland. Hepatic abscess and sarcoma of the thorax or abdomen may also give rise to febrile symptoms which are very obscure. Many a mysterious case of illness has been cleared up by a careful and elaborate examination of the chest, and the detection of a collection of pus. I need hardly go through the physical signs of empyema with you; I am sure you know them already. There are the

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signs of fluid in the chest, associated with a hectic temperature, fever, and wasting. Sometimes there is œdema of the chest walls, and sometimes in prolonged cases there is clubbing of the fingers. But the only absolutely certain way of diagnosing pus in the pleura is to pass in a scrupulously clean fine trocar and cannula, draw some of the fluid out, and examine it. If you are satisfied that there is fluid in the pleura, in cases of febrile temperature never delay about aspirating, and, as a rule, you will find pus, and thus a dubious case is cleared up. Now comes the question of operating in these cases. Supposing you tap a man's chest, and withdraw pus by the aspirator, you give him temporary relief. But in a few days he will be as bad as ever; the pus re-collects. It is necessary to make a free opening to evacuate it. I shall say a few words now about operation for empyema, because this may be a proceeding which every general practitioner may be asked to carry out. You will be obliged to make a free opening into the chest, and this operation may have to be carried out with poor assistance in a country village or an out-of-the-way place in the colonies. The usual practice is to make an opening fairly low down, at about the sixth or seventh rib, and excise a piece of one or two ribs, so as to allow very free exit for the pus. The seriousness of the operation is—and you will probably hardly credit what I am going to say—not so much in excising the ribs, for this can be done easily enough, but in giving the anæsthetic. The giving of an anæsthetic in a case of empyema is one of the most grave responsibilities that any medical man can undertake, and many surgeons, recognising this fact, do the operation with local anæsthesia, that is to say, by freezing the parts or injecting cocaine or eucaine.

But private patients can seldom endure such operations as cutting through the ribs with bone forceps unless they are to some extent placed under the influence of a general anæsthetic. The great risk with regard to the anæsthetic is this: there is only one lung at work, and if, as sometimes happens, a quantity of pus comes into the bronchus and flows down into the opposite lung, that lung also becomes choked, and in such a case the patient will, of course, at once die. I am sorry to say that amongst the unfortunate disasters from anæsthetics which I have seen in my professional experience, I know of two or three of exactly this kind. If the patient is turned over on the sound side, and the surgeon begins to operate, the risk is very great of pus flowing into the bronchus of the only working lung. Therefore the right position is that the patient be kept almost flat, with the chest drawn over the edge of the operating table, towards the side on which the operator is sitting, so that the diseased side actually projects over the edge of the table. The surgeon should perform the operation sitting upon a chair or stool, so that the side of the chest is in a dependent position, and the work of the opposite lung is not embarrassed. The anæsthetic employed should be either chloroform or the A.C.E. mixture. It must be very cautiously administered, and given in such a way that the patient is never very deeply under its influence. He should be able to make inarticulate sounds and move, and, perhaps, put his hand towards the site of operation, showing that the reflex actions are not entirely abolished. The patient should be barely conscious of pain; this is really a very important part of the whole procedure. If you place such a patient deeply under ether he is in danger. If you roll the patient over on his sound side the peril will be greater still. I have known the grave error committed of operating on the wrong side of the chest, an accident which proved instantly fatal. This terrible mistake may be quite avoided by a precaution which I am going to mention. It is this: no sensible man would open an empyema unless he saw the needle in position, and pus coming away from it. I make a rule never to believe what anybody else says about matters which involve issues of life and death. I never quite credit any one who may point at a mark on the skin and say, "That is where Dr. So-and-so put his needle in." Everybody may be inaccurate in these matters, and therefore the safe thing is to find the pus yourself, leave the needle in the collection, and follow the guide of this instrument along in the operation. By this means you are certainly led to the site of the pus, and you avoid the shocking error of opening the wrong side of the chest into a healthy pleural cavity. All these preliminaries having been arranged, you may turn up a large square flap, or make a cruciform incision, and rapidly bare the ribs of periosteum with a raspator. You cut out considerable pieces of ribs with the bone forceps,

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and with your finger or a director break through the thickened pleura into the abscess cavity. An enormous quantity of pus will gush out, and this may be terribly fœtid. A great deal of apprehension has been often experienced amongst doctors on account of the possible hæmorrhage in this operation and the bugbear of the position of the intercostal arteries. I have long ago ignored hæmorrhage in these cases. The intercostals are of small importance. Any actual "spurting" which takes place can usually be well dealt with after the ribs have been removed. It is easy to take half an hour or more over this operation if every little vessel is to be secured by catgut. Such a long time is occupied that the patient may well lose his life before the chest has been cut into. Patients with empyema are often desperately ill. It is very necessary that the operation should be done quickly. It should never take much more than five minutes, and this should include putting in the tube. These patients are too weak and collapsed to be kept for a long time under an anæsthetic. Do not wash out the pleural cavity at the time of operation, however foul the discharge may be. Very serious symptoms of collapse sometimes supervene if this is done. A tube is next introduced, and the tube which is ordinarily employed is, I think, much too small. To put in a tube of the diameter of a cedar pencil is absurd. The tubes I use for draining an empyema are quite half an inch in diameter. Through such a tube as this the accumulated pus can flow freely out. And now we come to a most vital point, and that is the proper securing of the tube. Anybody who has followed my practice will know how cautious I am in this respect. The tubes easily disappear within the chest, a grave embarrassment. The instrument makers sell tubes with india-rubber flanges, upon which they are supposed to be secured. The flange gets in time softened by the pus, and the tube slips into the chest. There is nothing more disastrous in a private case than for the nurse to take off the dressings in the morning and say, "Oh, the tube has gone inside!" I cannot warn you too strongly with regard to this matter. I have in hospital practice, once in this hospital, and several times in others, resected ribs to extract pieces of tubes. It is no fancy danger, but a thing which very readily occurs. Therefore to secure the tube you should attach to it three or four pieces of long strong silk, which you pass round the chest wall, and fasten on the other side with strapping, so that in case the tube slips in you can easily draw it out again. By taking such precautions this disaster cannot happen. I make it a practice never to bandage on dressings in these cases. They become sodden with fœtid discharge almost at once, and need constant changing. I like these patients to lie upon a large pad of "wood wool," and this as it gets sodden is pulled away with forceps and burned, and fresh wool substituted. The material should be lavishly used, and thus the patient is kept "dry" and comfortable. I have always thought, too, that the free movements of the chest greatly assist the expulsion of discharge. In about fourteen days I carefully begin washing out of the cavity. I would not dogmatise as to the agent employed. Dilute iodine and water is a very useful lotion, and so is peroxide of hydrogen. I will now pass on to consider the course of these cases. Many of them I fear go to the bad in hospital practice. They pass from one institution to another with a discharging and unhealed sinus. It is one of the many imperfections of hospital practice that we do not follow out the after-results of our operations, as we are bound to do in private. In hospital practice the patients leave, and in the notes you will find that the patient is "improved," or that the discharge is "diminished," or that he has left for a "home in the country," but seldom will you find that he is cured and sound. But in private practice these patients must be made absolutely well, and here comes the difficulty. You will find my words on this point very true, though you may not realise it now. Far and away the best chance of getting a case of discharging empyema well is to treat it by the seaside or in mountain air. The ideal treatment of discharging empyema is to expose the patient to sea air continuously, and for this purpose you cannot have better places than Margate and Ramsgate or Cromer in the autumn; indeed, any places along the East Coast. The air of these places works marvels, and I believe, on the whole, that Westgate or Ramsgate is the best. In the summer time the patient should dwell in a tent facing the sea, the sinus should be washed out with iodine and water, and this treatment will achieve wonderful results. In addition, of course, the patient should have good

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food, and plenty of it: "stout," fish, and tonics, and everything which will sustain and increase his strength. But you may have a case of this kind under treatment for three or four months, and still the sinus does not close. When the tube is taken out the patient begins to get fresh fever, and out comes another "burst" of pus. What is to be done in a case of this sort? There is only one thing to be done, and that is free resection of the chest wall. This is a rather formidable affair. It consists practically in removing enough of the bony framework of the cavity, for the chest wall to sink in. The operation becomes absolutely essential in many of these cases, but it should only be done under the best circumstances of nursing and dietary, and especially good air. I did this operation for the man in Hunter Ward; it has benefited him but little. He has no power of repair at all, and the opposite lung is full of tubercle.

A large number of cases of empyema which are met with occur in young people or children, and in them the conditions are more favourable than in adults. By removing a portion of rib, and exposing the child to the fullest extent to the influence of sea air, a cure will usually be effected. Healing is rapid, and the yielding chest wall soon contracts. A common mistake here is to take out the tube too soon, and allow reaccumulation of pus. But in a case of empyema in an adult, even if you can give it every advantage of sea air and the best of nursing and surgery, it is well to warn the friends and relatives that the course of the case must be prolonged, and the end dubious. But the results of those cases, seen in broken-down hospital patients, who cannot get the benefits of good air and all I have indicated, must not guide you too far towards a feeling of despondency in estimating the results in the better classes.

Next I shall mention a case which many of you have seen, and which some of you have been concerned in dressing, namely, a desperate instance of lacerated wound involving the knee-joint, in the Oxford Ward. The patient is a man æt. 28, who slipped between a railway train and the platform, and as a result got a bad injury to the knee. The soft parts on the inside of the joint were crushed and rasped away, exposing the interior of the joint freely, and laying bare the upper part of the tibia and cartilages. Such an injury in the olden days, and not very long ago either, would have been condemned to amputation as hopeless. Nevertheless this man's limb was saved, and his case affords a useful illustration with regard to a general statement which I shall now make. There are hardly any wounds of joints in these days which are so desperate as to need amputation. You may reflect in this hospital how seldom you see amputations. When I came here as a student amputation was one of the stock operations, and it was almost weekly performed. At first sight it looked almost like madness to try and save this man's limb. The treatment adopted in the case was as follows:—The limb was fixed upon an immovable splint. The best splint, I have no doubt, for fixing the knee-joint in bad wounds and injuries is that which Mr. Howse, of Guy's Hospital, invented, and advised for excision of the joint. It extends from the buttocks to the foot, and the limb is fixed in it by means of wax bandages. Dressings are put on over the splint, and they can be changed without that mischievous and painful lifting of a limb about, which is so deleterious and inimical to sound healing. It is a most admirable apparatus for injuries of the knee of this kind. If any of you in the future have a case of bad wound of the knee and want a splint to fix the limb, procure at once one of these splints. When the limb is fixed it may be "slung" in a Salter's cradle, to the great convenience and comfort of the patient.

But the first consideration is the proper cleansing of the joint. In this case we had the joint carefully flushed out and examined. And here came a discovery of great importance. Wedged into the popliteal space was a dirty piece of wood about three inches long. It was only by careful examination of the joint with the finger that this was detected. I may mention that I have known several bad disasters to occur in joint injuries from leaving a foreign body in the joint. I knew the case of a young man who was engaged in a "paper-chase," who sprang from a hedge into a ditch in which were some old stakes. He had as a consequence a lacerated wound which involved the ankle-joint. The surgeon who attended him very pardonably washed out the joint and united the wound. But the limb swelled up, and gangrene ensued, and the ultimate end was amputation. After amputation the foot was examined, and it was found that there was a piece of dirty wood in

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the ankle. This having been discovered, the parents of the youth attributed the unfortunate end of the case to carelessness on the part of the surgeon. A very unpleasant controversy ensued, the relatives persisting that the amputation was due to the doctor's carelessness or omission, and the matter was with difficulty kept out of the law courts. Therefore, in these cases always give a general anæsthetic, both for the purpose of properly cleaning the joint, and also passing the finger into it to make sure there is not a piece of stone or metal or wood embedded in the tissues. Our patient was anæsthetised, the crushed and torn tissues and the fringes of lacerated fascia were cut away with scissors, and the finger was passed freely into the joint. Then this piece of wood was fortunately discovered and extracted. The joint was repeatedly washed out. I saw the case was a very bad one, and I irrigated the wound with a solution of biniodide of mercury of the strength of 1 in 500. It was represented to me that a lotion of that strength was dangerous to the tissues, and that mercurial poisoning would likely occur. But although I freely irrigated the joint with this strong solution for many weeks, the patient never had any mercurial poisoning at all. The patient got little or no febrile temperature or pain, the wound granulated up, the exposed cartilage became covered with granulations, we put on skin grafts and they rapidly grew, and in about three months this apparently desperate and hopeless injury was recovered from. But what was the condition of the joint? As was expected, it was absolutely ankylosed. There was fibrous ankylosis, and ultimately bony ankylosis will ensue. This is the best result you can hope for in a case of injury of the knee-joint of such great gravity, where the soft parts are torn away, and the cartilages exposed and partially destroyed. But you will find the majority of mankind naturally object to stiff knee-joints, and the patient will complain of the stiff knee, and may even blame you for a result which is most fortunate, and the best that could have been obtained. Unjust blame and unjust praise are found in all branches of life. If you take the case of an unsuccessful general who loses a battle, everybody, with pipes in their mouths, sitting in their armchairs, will talk freely about it, and tell you how much better they could have done it themselves. And so when a surgeon has an unsuccessful operation, or a stiff joint results from his treatment, the relatives will blame the surgeon for it, say it was all his fault, and call him a blunderer or a fool. But you have to remember that this kind of thing happens to all men in public positions, and although it may vex a young man in early practice, he will soon cease to consider it. You will all get your meed of blame which you do not deserve, but you will also get your share of praise which you do not deserve, and so the two things will balance each other. Your duty in such a case as we are considering is to tell the friends and patient that there will most probably be a stiff joint for life, that there is no way of avoiding such a condition. If they choose, now is the time, early in the case, to call in other advice. Thus after-blame is avoided, because the result of a stiff joint is anticipated. The case we have treated in Oxford Ward was a desperately bad wound of the knee resulting in ankylosis. But many wounds of joints are not so bad as this. For instance, take a wound made with a clean knife; if such a case is properly treated you hope to get a movable joint afterwards. Such a result is usual after clean incisions in surgery. The treatment is to wash the articulation out carefully with strong antiseptic lotion, unite the wound most accurately, put a tube in for twenty-four hours, and as soon as the wound is soundly healed begin passive motion. In a young, healthy individual, in a case like this you will certainly get a good joint. When speaking of wounds of joints nearly everybody instances the knee. Of course the joint injured usually is the knee because of its exposed position. But there are other joints which are quite commonly the seat of wounds, and we are apt to ignore them; I mean the finger-joints. Remember that a joint of the finger is liable to the same diseases and injuries as joints elsewhere, and if such a joint is affected by septic inflammation there is destruction of cartilages and disease of the synovial membrane and deformity. And, indeed, such a condition in a finger-joint is a very serious matter. A dirty nail may prick a finger-joint. In the case of fishermen or cooks, the spines of fish may wound one of these little joints. Or trouble in a finger-joint may result from the too free surgical treatment of whitlows. In the "too free" opening of whitlows the point of the knife may just prick the joint, and let the pus from the whitlow into it. These septic wounds of

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the fingers are very serious, and they often result in ankylosis of joints.

Wounds of joints, especially of the knee, shoulder, and elbow, may occur from an injury at some distance from the articulation; this is especially the case in punctured wounds. A man falls from a hayrick, for instance, on to a pitchfork, and you will find a wound in the thigh, perhaps six or seven inches from the knee. But be careful that the sharp instrument has not run down under the skin and muscles and wounded the synovial membrane of the knee-joint. If this has happened you find the joint swollen, and you can generally squeeze synovial fluid out of the wound on pressure. In such a case as this I should not hesitate to lay open the wound, and if there was effusion into the joint to open the articulation and wash it out with strong biniodide of mercury lotion. These are only some of the many practical details in connection with wounds of joints. They are common accidents of civil life, and you may all meet with them in future practice.

I will next say a few words about an elderly man who was in the Grosvenor Ward for some time with a common and important complaint, namely, tuberculosis of both testes. This is a remarkable case, because tuberculosis of the testis is so commonly a disease of young adult life; it gets rarer as life advances. But still some cases are found in persons of advanced age. This patient was close on sixty years old, and had suffered from the malady for a year.

As I have pointed out to you constantly in the wards, tuberculosis of the testes in elderly men runs a very chronic course, and it is seldom necessary or advisable to remove both testes, as is, I think, too often advised. These cases in old subjects are usually to be treated by opening the abscesses as they arise, scraping out the caseating foci, and dressing the sinuses with iodoform gauze. This treatment, combined with sea air and tonic treatment, will usually result in, not cure, because such is hardly the word to use in this connection, but in a complete amelioration of the symptoms. The abscesses will discharge, and the testes will shrivel up into fibrous masses, containing, perhaps, but little useful testicular substance, but such a condition will be sufficient to keep the patient free of that mental trouble so often seen in cases of double castration. I hold the view, I fear now antiquated, that the removal of ovaries in the female, or of both testes in the male, are very serious measures to adopt. Double castration for enlarged prostate will have its day. It has not come to stay with us. Removal of both testes for tubercle should be avoided if possible; if any other operations can be substituted perform them. The efficacy of good sea or mountain air in these cases is surprising, and should ever be remembered before recourse is had to such advanced surgery as double castration.

The next case I draw your attention to is one in the Wellington Ward, a typical instance of that terrible disease known as acute diffuse periostitis, or, what is perhaps a better name, acute necrosis of bone. This is a very remarkable disease. I will say a word about its pathology. It is dependent in some obscure way upon the entrance into the tissues of septic organisms (streptococci probably), into the canals of the bones and periosteum, setting up a localised violent septic inflammation, leading to choking of blood-vessels and the rapid death of the bone. It is a very remarkable disease, because the question arises, How do these organisms enter without wound or breach of surface? They are said to gain access to the body in many ways, but I favour the idea that they enter through the throat. Thus the malady is found as a sequel of the specific fevers, especially scarlet fever, when the throat is unhealthy and ulcerated extensively. The exciting cause is almost always a blow, often a slight blow, or exposure to cold, and then the resistance of the tissues being lowered, the organisms collect, proliferate, and produce local suppuration and death of tissues. The exposed bones are usually the site of this terrible malady, and in the vast majority of cases the bone involved is the tibia, but I have also seen the femur, the humerus, the clavicle, and the sternum implicated. The symptoms are as follows:—Almost invariably the patient is young; I have never seen a case in adults. The age is generally about twelve or fourteen, at all events under twenty. He is smitten with very acute local pain, high fever, and the neighbouring joint very often swells. And here I must note a lamentable mistake which is often made in these cases. This swelling of the joint invariably creates in the minds of some doctors the impression that the condition is rheumatism, which, like the cloak of charity, covers a multitude

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of wrong diagnoses. We are all too apt to use the term rheumatism in connection with any pain the nature of which is dubious. Several joints may be swollen, the effusion being really pyæmic. The diagnosis from "rheumatism" is really absolutely clear, because there is no acute rheumatic affection which ever causes acute inflammation of the shafts of the bones. If you find the shaft of a long bone tender and inflamed, with acute pain and local redness, you may be sure it is something more than rheumatism. The parts become red and glazed, and the temperature reaches a great height; the patient has rigors, he is in intense agony, and it is necessary to give morphia liberally to keep him quiet. Thus the symptoms go on; the tongue becomes dry and cracked, the cheeks deeply flushed, the patient very ill and prostrate. Then when an incision is made out comes an enormous quantity of yellow pus, swarming with staphylococci and streptococci, and the whole of the bone is seen to lie bare and dead, and bathed in pus in a cavity bounded by the thickened and receding periosteum. Other conditions are often associated. I have spoken of pyæmic effusions into the joints. With the stethoscope you hear a pericardial rub. Here is another way in which diagnosis may easily err: you put your stethoscope over the pericardial region and hear a friction sound, and at once conclude it must be "rheumatic." As a matter of fact, it is a serious pyæmic inflammation of the pericardium. These patients often pass blood in their water, and this symptom in the case puzzles the doctor beyond belief. The explanation is simple enough: pyæmic infarcts in the kidney, with intense local congestion. *Apropos* of the treatment of this formidable disease, first of all, do not mistake it for rheumatism. If you do so, and tell the parents that a favourite child has rheumatism, that you will give some medicine, and that he or she will be all right in a day or two, and there follows an acute necrosis of the tibia, with a year's illness, you will not be highly thought of for the future in that family. Next, as early as possible cut down upon the affected part, and if you operate in time, trephine the bone as well as cut through the periosteum. By this means you relieve the tension, and you may possibly limit the necrosis. It is stated that early trephining will cause recovery. I have never seen a case where necrosis has not occurred. By the time diagnosis is clear the inflammation has far advanced. In the majority of cases the whole shaft of the bone dies. When this eventuates there are two things to be done: you may make a long incision and remove the whole bone, allowing the periosteum to form new osseous substance, or you may, after the old fashion, allow the periosteum to throw out a shell of new bone, and in a year or two years afterwards cut through the shell and remove the sequestrum from within it. I hardly know which is the best practice to adopt. In the last years I have taken out the bone at once in several instances, and I think I shall do so in the case now in the wards. In the last case I had in this hospital the upper epiphysis of the tibia was affected as well, the knee-joint was destroyed, and amputation became needful. This disease almost always stops at the epiphysial lines, because the epiphyses do not receive their blood-supply from the same source as does the shaft of the bone. In cases in which the epiphyses are attacked the joints generally become hopelessly disorganised.

I have only mentioned a few of the many cases of interest now in the wards of this hospital. The lesson teaches what I have often maintained, that the study of six cases is better than the perfunctory observation of fifty. The best "clinical laboratories" are the wards. There is a sad tendency now to substitute elaborate methods of clinical investigation carried out in laboratories for bedside observation. Perfection in the use of the stethoscope in percussion, and in the hundred minutiæ of bedside observation, will alone make a good practical doctor. And the public are good judges of these points, for the man who neglects them need not have care or anxiety about the cure of his future patients; he will have no patients to cure.

Phosphorus Treatment of Rachitis.—Leo ('Jahrbuch für Kinderheilkunde,' vol. liv, No. 5) saw two fatal cases of phosphorus poisoning in rachitic children treated with phosphorus during the past year. One case took cod-liver oil and phosphorus for fifteen days, receiving in all 0·015 of the latter. The other child had been given 0·001 of phosphorus daily for almost six months. The symptoms which preceded death were icterus, apathy, and, in one case, swelling of the liver.—*Amer. Journ. of Obstet.*, April, 1902.

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HYPNOTISM:

AN OUTLINE SKETCH.

Being a Lecture delivered before the King's College Medical Society.

By J. MILNE BRAMWELL, M.B., C.M.

HYPNOTISM is too vast a subject to be condensed into the compass of a single lecture. I propose, therefore, to give you an outline sketch of its rise and development, in the hope of arousing your interest and stimulating you to study the subject on your own account.

Just as chemistry arose from alchemy, astronomy from astrology, so hypnotism had its origin in mesmerism. Phenomena, such as Mesmer described, had undoubtedly been observed from an early date in human history, but to his work, which extended from 1756 to his death in 1815, we owe the interest that was excited in the scientific world, and which led, after the exposure of much error and self-deception, to what we now term hypnotism.

Possibly most of you have heard of the hypnotic work of Charcot and his followers, and of their belief that metals, magnets, drugs in sealed tubes, etc., exercised an extraordinary effect upon hysterical subjects. Further, how Liébeault, of Nancy, and his disciples claimed to have demonstrated that the effects just referred to were entirely the result of suggestion. I question, however, whether many of you are acquainted with the fact that the rival schools of the Salpêtrière and Nancy had their counterpart in England sixty years ago in those of the mesmerists and hypnotists; that the fallacies of the Salpêtrière were simply the revival of old mesmeric ones which Braid had successfully combated, and, further, that he employed suggestion for the production of hypnosis and its experimental and therapeutic phenomena, just as the members of the Nancy school now do; finally, that his ideas of the conditions involved in suggestion were more scientific than those held on the Continent at the present day.

John Elliotson, the foremost physician of his time, was the leader of the English mesmeric movement just referred to. In 1837, after seeing Dupotet's mesmeric work, he commenced experiments at University College. By means of monotonous passes and other physical methods, he produced a species of trance in many subjects, frequently accompanied by anæsthesia profound enough for operative purposes. This form of treatment was often followed by the relief or cure of various diseases, particularly functional nervous ones, the curative effects of the trance being apparently increased by the influence of magnets and various metals, which also seemed to be the exciting causes of the many strange mental and physical phenomena presented by the subjects. Students from all parts crowded to Elliotson's lectures, but these had soon to be abandoned. The Council of University College forbade the employment of mesmerism, and, in consequence, Elliotson indignantly resigned his professorial appointments, and quitted for ever the hospital he had done so much to establish.

The action of the Council was largely due to the hostility of Elliotson's colleagues, who had long resented his unorthodox ideas and practice. Indeed, he held more dangerous and revolutionary opinions than I can refer to in detail. In his earlier days he condemned the universal practice of blood-letting, and asserted that thousands of persons were killed by it, while in his later ones he complained that a remedy, which undoubtedly was useful in certain instances, had been completely abandoned.

He carried on many therapeutic experiments, and pointed out the value of quinine, iodide of potash, hydrocyanic acid, creosote, and other drugs which were little thought of at that time. In addition, he attacked the manner in which drugs were generally administered, and asserted that many medical men had actually a fixed dose for each medicine, while others changed their prescriptions incessantly, before their drugs, either from smallness of dose or shortness of the period of administration, had had the least chance of doing good.

Elliotson's interference with blood-letting and methods of drugging gave great offence, but it was something entirely different which, in the opinion of his colleagues, stamped him as quack and charlatan. He actually began to carry a piece of wood about with him; this he called a stethoscope, and asserted that, by placing one end of it to the patient's chest and the other to his own ear, he could detect different sounds in connection with the heart and lungs. Naturally other teachers of

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medicine treated all this with indignation or contempt, and described Elliotson as a fool who carried a piece of wood into a sick chamber and indulged in idiotic hocus-pocus with it.

Elliotson continued his mesmeric researches up to the day of his death, despite the persistent and virulent attacks of the medical press, which would have ruined any one of smaller fortune and less powerful personality. In 1846 his turn came to deliver the Harveian oration, and, as soon as it became known that he had accepted the office, he was attacked in the most savage manner in order to prevent his appearing. Undeterred by this, he made mesmerism the subject of his address. Without saying a word as to the attacks which had been made upon him, he simply stated the result of his researches, and most respectfully invited the College to examine alleged facts of overwhelming interest and importance. His speech is too long to be quoted in detail, but he exhorted his hearers to study mesmerism calmly and dispassionately, and reminded them, with more truth than tact, that all the greatest discoveries in medical science, and the most important improvements in its practice, had been opposed by the profession in the most violent and unprincipled manner. As examples of scientific discoveries which had been received in this way, he cited those of the lacteal vessels, the thoracic duct, the sexual system of plants, the circulation of the blood, the sounds of the chest and their relation to the diseases of the heart, lungs, and their coverings, etc. As instances of improvement in practice which had been treated in like manner, he referred to the employment of Peruvian bark, inoculation and vaccination for smallpox, the use of mild dressings in gunshot wounds instead of boiling oil, and the ligature of the bleeding vessels after operation, instead of the application of burning pitch or red-hot irons. In conclusion, he urged his hearers neither to forget these facts nor to allow authority, conceit, habit, or the fear of ridicule, to make them hostile or even indifferent to truth.

As a result of Elliotson's influence, mesmeric treatment was extensively and successfully employed in many parts of the country. All who meddled with it, however, were the victims of incessant professional abuse and persecution. Medical societies rarely allowed them to record their doings, and when they did, treated them with scant courtesy. Thus, Mr. Ward, a Nottinghamshire surgeon, amputated a thigh during mesmeric trance; the patient did not make a single muscular movement during the entire operation, and was afterwards unable to recall the slightest sensation of pain. The case was reported to the Royal Medical and Chirurgical Society, but was badly received. One of its members, Dr. Copland, proposed that no account of such a paper having been read before the Society should be entered in its minutes, as pain was a wise provision of Nature with which it was impious to interfere. He asserted that patients ought to suffer pain during operation, as this did them good and enabled them to recover better.

The fame of Elliotson's mesmeric work reached India, and in 1845 James Esdaile, a surgeon in the service of the East India Company, determined to investigate the subject. At this time he was in charge of the Native Hospital at Hooghly, and had under his care a convict about to undergo a painful operation. Esdaile mesmerised him, and succeeded in rendering him insensible to pain. This encouraged him to persevere, and some months later he reported a number of successful cases to his medical board. His letter received no reply, despite the fact that up to this date no anæsthetic of any kind had been employed in surgical operations in India. At the end of a year Esdaile reported 120 painless operations to the Government. In consequence, a committee, largely composed of medical men, was appointed to investigate his work; their report was favourable, and Esdaile was placed at the head of a Government hospital in Calcutta for the express purpose of mesmeric practice. From this date until he left India, in 1851, he occupied similar posts. He recorded 261 painless capital operations and many thousand minor ones, and reduced the mortality in the removal of the enormous tumours of elephantiasis from 50 to 5 per cent. Patients flocked to him from all parts of the country, and the record of his painless mesmeric operations forms one of the most fascinating and romantic pages in the whole history of medical science.

After leaving India, in 1851, Esdaile settled in Perth, and about a year later informed Elliotson that he found the inhabitants of the far north as susceptible to mesmerism as those of the farthest east. Dr. Fraser Thompson, one of the visiting

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surgeons to Perth Infirmary, became a convert and performed some successful mesmeric operations. His colleagues, however, called a meeting of the directors, and stated that they would resign if the practice of mesmerism were permitted in the hospital.

Fraser Thompson's experiments were afterwards reproduced by my father, the late Dr. James Paton Bramwell, of Perth. When a boy I witnessed many of these, and was deeply impressed by them; I also eagerly devoured such books on the subject as my father possessed, notably Dr. Gregory's 'Animal Magnetism' and a translation of Reichenbach's work.

While Elliotson and his fellow mesmerists were fighting adversaries, who represented the cause of ignorance and prejudice—for many of them openly boasted that they had never seen a mesmeric experiment, and that nothing would induce them to examine the subject for themselves,—they were soon to be face to face with another and more formidable opponent. This was James Braid, justly termed the father of hypnotism, a Scotch surgeon practising at Manchester.

Braid commenced to investigate mesmerism in 1841, and is now only remembered in connection with it and hypnotism. Long before that date, however, his fame as a surgeon had attracted patients from all parts to Manchester, and he had already operated on twenty-three cases of spinal curvature, 262 cases of talipes, and 700 cases of strabismus.

At first he believed mesmeric phenomena were due to self-deception or trickery; further investigation, however, modified this view, and he became convinced that a large proportion of them were genuine, but that all were explained in a fallacious manner. After making a series of experiments, he successfully demonstrated that the phenomena were entirely subjective in their origin, and absolutely independent of any mysterious force or fluid emanating from the operator. Braid's scientific explanation dealt a death-blow to many fallacies and marked the birth of a new science, which he termed hypnotism, in contradistinction to mesmerism. From this date, at all events amongst scientific men who had studied the subject, mesmerism was entirely discredited, and the justness of Braid's observations admitted. In truth, however, Braid had not really destroyed mesmerism root and branch and substituted hypnotism, as something totally distinct, in its place. He had rather, as it were, taken over the business of his predecessors, written off as valueless a large proportion of what they regarded as their assets, and reconstructed a company on new lines. Thus, amongst hypnotic phenomena are to be found such mesmeric ones as have stood the test of rigorous investigation, and are now explained in a different and more scientific way.

The following are the principal points on which Braid and the mesmerists differed or agreed:

1. According to the latter the mesmeric state was induced by physical means, notably monotonous passes with contact.

2. In Braid's opinion the condition could be invoked by verbal suggestion, without the employment of any physical means whatever; while the latter were invariably without effect, unless the subjects knew and expected the results which they were supposed to bring about.

3. Mesmeric phenomena, supposed to be due to the action of metals, magnets, etc., Braid explained by unconscious or indirect suggestion on the part of the operator. The phenomena themselves he still regarded as genuine, whether physical ones, such as catalepsy, or psychical ones, such as hallucinations of the senses, as, for example, when the patients believed they saw lights streaming from the poles of the magnets.

4. Braid held that the alleged phenomena of thought-transference and clairvoyance were due to unconscious suggestion or self-deception on the part of the operator, and hyperæsthesia of the subject's special senses.

5. Subject to the limitations just referred to, Braid regarded the so-called mesmeric state as a genuine one. As we have seen, he explained its origin differently, and called it the *hypnotic* condition or *hypnosis*, regarding it as an artificially produced sleep.

6. Later he observed that only 10 per cent. of his subjects passed into a state even superficially resembling sleep, and believed that the condition was due to an involuntary concentration of the attention. He proposed, therefore, that the term hypnotism should be abolished in favour of that of *monoideism*.

7. Finally, he recognised that volition remained intact in hypnosis, and that the attention could be

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directed to several points simultaneously; thus, the condition had nothing in common with involuntary monoideism or automatism. He observed also that, except when specifically inhibited by suggestion, consciousness was unimpaired. He recognised the alternating memories presented by deep hypnosis, and was impressed by the fact that the subjects in the normal state remembered nothing of what had happened in hypnosis, but, when re-hypnotised, recalled both the events of waking life and those of previous hypnoses. Having discarded the terms mesmerism and hypnotism, he finally rejected monoideism in its turn, and called the condition *double consciousness*. He contemplated publishing a full account of his latest theories, which apparently sought to explain the phenomena of hypnosis by the intelligent action of a secondary self, but his death occurred before he was able to carry out the project.

In 1843, less than two years after he commenced his researches, Braid published 'Neurypnology, or the Rationale of Nervous Sleep.' This work, which forty years later was translated into French, was long the only one of Braid's writings with which even the students of hypnotism were acquainted. Braid, however, carried on his hypnotic work for seventeen years after its publication; during that time his views underwent complete change, and it is in his later writings alone that is to be found a clear explanation of the doctrine of suggestion, together with reference to the theory of double consciousness. In 1896 I published an account of Braid's writings, of which at that time I had been able to trace thirty-two, a list I have since considerably increased.

Braid frequently used hypnotism as an anæsthetic, and with its assistance performed a number of painless minor operations.

In a case of spinal curvature, which had resisted ordinary treatment for six years, Braid obtained rapid improvement by removing all mechanical apparatus, and rendering the morbidly weak muscles cataleptic by suggestion. He believed he could cure recent cases in this manner without pain or inconvenience to the patient, and considered mechanical supports, except where there was actual disease of the bone, worse than useless, as they favoured the weakening of the muscles.

Braid employed hypnotism with much success in his medical practice; his most brilliant results were obtained in cases of hysterical paralysis, and in many of these all ordinary methods had entirely failed. He was also successful in treating other forms of hysteria, as well as epilepsy, neuralgia, chorea, dipsomania, obsessions, rheumatism, dyspepsia, constipation, menstrual disturbances, certain forms of deafness, etc. He even succeeded in relieving and improving many cases of paralysis of organic origin, and demonstrated that the symptoms in such cases were not entirely due to the extent of organic injury, but were also in part the result of functional nervous disturbance arising from, and associated with, the organic cause. He thus anticipated the observations of Charcot and Bernheim.

Braid also reported an interesting case in which he succeeded by suggestion in augmenting and maintaining the secretion of milk.

At first Braid employed certain physical methods; he soon recognised, however, that these acted entirely through the mind, and were, in fact, a form of indirect suggestion. He therefore abandoned them, and employed direct verbal suggestion alone, both for the induction of hypnosis and its experimental and therapeutic phenomena.

Liébeault and his followers have reproduced—without improvement of any kind—Braid's methods of inducing and employing hypnotism. The difference between them is not to be found in the way they use suggestion, but in the value they attach to the term itself in its relation to hypnotic theory. To this point—one too complicated to be discussed in detail—I will again, though shortly, refer.

Notwithstanding the justness of Braid's views as to the subjective origin of mesmeric phenomena, and the attention drawn to his theories and practical work by such well-known men of science as Professors Carpenter and John Hughes Bennett, the practice of hypnotism apparently ceased in England after his death in 1860. It was due to Braid's influence, however, that my attention was again drawn to hypnotism. In the physiological class room at Edinburgh University, I was fascinated by Professor John Hughes Bennett's account of Braid's work and theories, and impressed by his advice that we should never lose interest in a subject, which one day would revolutionise the theory and practice of medicine.

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Soon after leaving Edinburgh I became busily engaged in general practice, and hypnotism was almost forgotten, until I learnt that it had been revived in the wards of the Salpêtrière. I knew little of the methods employed there, but determined to go to Paris to study them. Before I was able to do so, a case occurred in my own practice which apparently required hypnotic treatment. The patient, an educated man, markedly strumous, suffered from urinary fistula, the result of an accident. When I first saw him there was a large unhealthy wound from which the urine escaped. I instructed him to pass a soft catheter, and matters improved, but shortly afterwards a mental condition arose which seemed to render recovery impossible. The idea of urination was immediately followed by an involuntary contraction of the bladder, whereupon the urine escaped from the wound before the patient had time to pass the catheter. This appeared to be entirely independent of the physical condition of the bladder, which did not contract because it was full or uncomfortable, but because the idea of urination presented itself to the patient's mind, and was instantly translated into its physical equivalent. Although I told him that I knew very little about hypnotism, I had no difficulty in inducing hypnosis. I repeated the process on the two following days, then suggested that he should cease to think about his bladder, should always be able to pass his catheter, retain his urine eight hours, and sleep well. These suggestions were fulfilled; from that day there was no return of his troublesome symptoms, and the wound healed without operation in about twelve months.

I then tried hypnotic treatment cautiously amongst some of my patients whom I had failed to relieve by ordinary methods, choosing them chiefly from amongst friends, and exacting secrecy, as I knew what had happened to other pioneers.

Lack of time prevents my giving details, but amongst the more interesting ones may be cited cases of pruritus vulvæ and eczema, hyperidrosis, long-standing headache associated with ciliary spasm, dipsomania, amenorrhœa, etc.

Success made me bolder, and I hypnotised many of my patients on my rounds and left them quietly sleeping, while others were treated collectively at my own house. In the latter case, I rapidly hypnotised a number of patients one after the other, gave them appropriate suggestions, and left them to repose for an hour or so before arousing them.

Hypnosis was rapidly induced in most instances; in 500 cases I only failed twice.

I found that suggestion often relieved or abolished pain, enabled patients to secure sleep at will, to escape from obsessions, conquer the diseased craving for alcohol and narcotics, and get rid of numerous functional nervous disturbances.

In many instances I induced hypnotic anæsthesia by suggestion, and made use of this for minor surgical work. Thus, in March, 1890, the late Mr. Turner, of Leeds, performed a number of dental operations upon my patients, which he recorded in the 'Journal of the British Dental Association.' The most remarkable case was that of a young girl, suffering from valvular disease, from whom he extracted five teeth.

On March 28th, 1890, a number of my patients were operated on at Leeds, in the presence of some sixty medical men, including such well-known surgeons as Pridgin Teale, Mayo Robson, etc. A delicate girl was put to sleep by written order while I remained in another room, and sixteen teeth were extracted. She showed no signs of pain, there was no corneal reflex, and the pulse fell during the operation. A boy æt. 8, suffering from exostosis of the great toe, had only been hypnotised on two previous occasions. Mr. Mayo Robson first performed evulsion of the great toe-nail, then removed the bony growth and part of the first phalanx. Some of the patients were strong healthy labouring men, others were weak nervous women. None suffered from pain afterwards, and all returned home by train—a journey of over an hour—while in every instance the healing process was remarkably rapid. An account of these operations was published in the 'British Medical Journal' and the 'Lancet.' Professional interest was excited, and many patients were sent to me from different parts of the country. The majority of them suffered from grave disease, and had been rendered hopeless by the failure of long and varied treatment. In most instances the induction of hypnosis was difficult and sometimes impossible, while the deep stages, characterised by anæsthesia, were rarely reached. Despite this, good results were frequently obtained. I determined, however, to visit hypnotic centres abroad in order to study the difficulties encountered by others, and their methods of combating them.

(*To be concluded.*)

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SOME PASSING THOUGHTS ON BROAD LINES OF TREATMENT.*

By W. J. TYSON, M.D., F.R.C.P.,
Physician to Victoria Hospital, Folkestone.

After some twenty years of practice, hospital and private, it is interesting to me, and I trust it will be so to you, to reflect on some of the numerous and various changes that have taken place in the treatment of disease, and to notice one's own attitude towards these changes. Treatment, both medical and surgical, has undergone within a few years an almost revolutionary change; twenty to twenty-five years ago cases of appendicitis were rarely, if ever, operated on, ear surgery as it is known to-day scarcely existed, gynæcology was just beginning to be studied seriously, stones in the bladder were cut out, but not crushed, operative suppuration was seen every day, and so I might go on. Specialities have grown and developed in every direction, and to-day there is hardly an organ in the body without its special attendant.

We must admit, I think, that a large proportion of the great advances and discoveries in treatment have been brought about by men engaged in special work, and at times when they had leisure to digest their thoughts and their methods; it will suffice to mention the treatment of the diseases of the eye, the ear, and the throat to illustrate this fact. But as at one time we were too general and too inexact, so now I venture to think there is a danger of being too specialised. Specialism seems almost rampant, and there is a serious danger of the general man being held of slight account.

There is another subject closely allied to treatment, viz. the age and the experience of the practitioner. The enthusiasm and immense energy of youth seems to require an outlet, but the reading it undertakes cannot as a rule be accompanied with much practical experience. The young man is certain and dogmatic in his diagnosis and treatment, while the old man is apt to be desultory and too hesitating—the young man thinks often that his senior is slow and timid, while the elder man forgets the advantage of youth; both at times are right, and both at times are wrong. I should say roughly that operations are more easily, and with less indecision, undertaken in early than in later life; at the same time the well-known operations which are beneficial would be more earnestly recommended by men of a few years' standing. There are a great many cases in which there is no doubt as to the value of operating; there are others, perhaps not as many, in which it is very difficult often to decide as to the necessity of interference. Here I would mention the question of hospital and private cases. We must acknowledge that we at times decide as to our treatment whether the patient is at home or in hospital—the difference between operating in a well-equipped hospital and a squalid, insanitary room is so great that it is no wonder to me that men often refuse to operate when the patient is found in the latter position. But to return to my point of age, there is no doubt that as we get older we think more and read less, or shall I say rather that we think more about what we read and have read, and also bring into mind our past failures and successes.

There is a saying that at times one cannot see the wood for the trees—or, what is more to my purpose, I would say that a man may be too near the wood to see it—in practice we are constantly, so to speak, too near our cases, and a mistake we often make is prescribing for a case at a first consultation or visit; how often I have wished I had not prescribed! A first consultation is often well spent in hearing the patient's history only. Again, the patient often comes to be treated for a local trouble which has a distant cause, and to remember the various and multitudinous causes of it often takes more than five or ten minutes given to a case. I have often said one third of my eye patients that come to me require general treatment; let me give you an example out of many in this branch of medicine. A man æt. 54 came to me with sudden loss of vision of the right eye; on examination a large hæmorrhage was discovered in the posterior region of the eye. His history was this: that for years he had been subject to albuminuria, large hypertrophied left ventricle, and loud systolic murmur, with increased arterial tension, and other symptoms of general gouty degeneration. The man had always lived well, and had taken little or no exercise. He at once wished to consult an

* Delivered before the members of the East Kent District of South-Eastern Branch of the British Medical Association at Dover, March 27th, 1902.

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oculist, and saw an eminent one in London. It was apparent it was not primarily an eye case; the position of the hæmorrhage was accidental, and the real treatment of the case was general. This line, I need hardly say, the specialist took, and handed the case back to my treatment. To illustrate further let me take the treatment of eczema. I think that there can be little doubt that general causes play a very important part in the origin of the above disease; this fact was known to a past age of practitioners when they said that some forms of skin disease should not be driven in—by that they meant, I suppose, local applications were not always beneficial. I remember well my father rebuking me for attempting to cure locally a gouty scrotal eczema. I often have heard men laugh at the "driving in" treatment, but, taken in its true light, there is much sense in it. Still, local remedies hold an immense sway in the treatment of skin diseases; and in looking through my prescription book, under the head of eczema, I find many more local prescriptions than general ones. My present opinion is that internal remedies, if carefully selected in regard to the cause of the eczema, are, as a rule, of more use than local applications. The public like an external treatment in which they can take a prominent part and see what is going on, and I think that this is a reason which accounts partially for the present prevalence of these applications. I could quote many cases in which my patients have not done well until internal treatment was carried out.

Pharyngitis is another disease which seems to me to have been treated locally *ad nauseam*, with more often than not no corresponding beneficial results. I am now referring particularly to that chronic form which is called granular. The disease is often met with in delicate young women, and may or may not give rise to irritating symptoms. Every conceivable form of local treatment has been tried, but I suspect the electric cautery has had the longest innings. A case came under my care during last summer like one of those I have mentioned: the pharynx had been actively treated for years until she finally decided to think a little more about her general health, and it was then that she began to improve. Very often the change of food, suitable clothing, free ventilation, and regular exercise are almost too prosaic to mention, still less to urge to the notice of the patient, but until these broad rules are carried out the local treatment will have comparatively little effect. The cause of the pharyngitis may be a general and removable one, and until this is attacked the local applications only complicate and mask your treatment.

Again, I would refer to some interesting cases in which we are primarily consulted for difficulty of breathing, but which do not improve or get well until a general course of treatment is adopted. The cases I am thinking of occur both in women and men, in ages between 50 and 70. These patients are stout, indolent, well-to-do, and enjoy and partake of the good things of the table. On further inquiry you will find the shortness of breath is not permanent, and is often associated with a particular increase or unsuitability of food. On examination the left heart seems enlarged, impulse feeble, and the pulse is quick and weak; the bowels are constipated, and albumen may be present in the urine. They sometimes complain of tenderness and weight over the liver, and a general lassitude of mind and body; their nights are often sleepless or restless. On first sight one is tempted to give heart tonics and stimulants, and in small doses. I am not prepared to say that this is wrong, but under these without other means the patient does not improve. The diet requires very carefully to be regulated, food given in small bulk, solid rather than fluid, and about every three hours. The bowels must be kept free; perhaps the most valuable drug at this stage is calomel given in frequent and small doses. It is advisable for the first few days if possible to keep the patient in bed, as the free opening of the bowels may produce faintness and syncope if the patient is up and about. As soon as the patient is better moderate exercise should be insisted on, and a general plan of moderation in all things should be written down and pressed. The foregoing description will probably call up in your minds similar cases you have met with. I think that the breathing difficulty is brought about by the flabby condition of the heart setting up some dilatation, and this is followed by pulmonary and liver congestion, the original cause being the overloading of the abdominal organs from excessive or unwholesome foods, together with the absence of exercise or good air, and the inability to dispose of the waste products. At any rate my experience

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is that no amount of tonics, whether cardiac, nervous, or intestinal, have any real effect until the patient is put upon and acts upon a rational system. of living. I should not, perhaps, have alluded to these cases except I have made mistakes myself, and seen them in the practice of others, in which the value between the local and general troubles was not proportionately estimated.

There is a common malady named lumbago, with its ally sciatica. The causes of it given in our text-books are many, and popular remedies are numerous. To treat the complaint successfully the numerous causes must be borne in mind, and each considered in connection with the case under consideration. A belladonna plaster or an injection of morphia may be necessary as a crutch to the lame man temporarily, but in the vast majority of cases this cannot be put down as treatment. Sir George Johnson's term of muscular dyspepsia or dyspeptic myalgia as expressing a condition of the lumbar muscles, which are irritated and kept sore by the products of imperfect digestion or secondary assimilative processes, seems to me to be a far more common cause of the above than is usually thought or taught clinically. The many cases that get better when treated with anti-dyspeptics, cholagogues, and aperients go largely to support this view. It is very common to see the word rheumatic affixed to the word lumbago, and anti-rheumatic remedies are often prescribed; but I have sometimes thought that as many of these cases are now treated at inland health resorts, such as Buxton, Bath, Aix-les-Bains, etc., the careful dieting, massage, baths, alterative and aperient waters, all more or less affect the general system, and have an eliminating action, and the success of the treatment of lumbago at health resorts is largely due to what I have stated.

Let me give you one more prominent symptom, viz. convulsions, in which one must often look beyond the brain for the explanation and its treatment. Here I will shortly describe a case as illustrating the point I wish to bring forward.

A boy æt. 2 years was taken almost suddenly with severe convulsions; I saw the child soon after the convulsions had begun. These soon became almost continuous, and the boy appeared in a serious condition. On placing my hand on the abdomen marked peristaltic action of the bowel was felt. The convulsive attack was so tedious and violent in passing off that I gave some chloroform to check it. Some time later the bowels were opened four or five times, after which the convulsions entirely passed off. There are many causes of convulsions in children, and intestinal irritation from undigested or ill-assorted food is among the most common.

In regard to the convalescent stage of disease my opinion, from what I have seen in my own practice and in that of others, is that not infrequently patients are ordered to work before the health is fully established. The obvious answer to this statement is that in most cases time is too precious for the patient to spend very long in convalescence; but we must remember that convalescent homes are comparatively common, and admission to them by the poor is not difficult, and the other classes often have opportunities, or could make them, for a longer rest after illness if the medical adviser urged it.

It is only lately that I have fully realised how tedious is a full recovery after many cases of typhoid fever, and I will quote this disease as an example of many cases in which prolonged rest is required for recovery. What a serious exhaustion to the whole nervous system a case of typhoid must involve. A fever, almost continuously high, lasting often for four or five weeks, with a great portion of the bowel out of gear, all the internal organs more or less softened and worked at the greatest disadvantage. Is it a wonder that the convalescence must be and ought to be slow? The seriousness of the attack is seen later on in many ways, such as the loss of hair, the formation of nodes or abscesses, the feebleness of the muscular system, the slowness of the digestive organs to re-establish themselves, and the mental weakness or defective memory, etc.

It is a hurrying age, and everything must be done quickly, and doctors like other people have fallen under this stress of example. Of course, there are many exceptions to my statement; every case must be considered from what the disease is, and also the quality of it. I have made more mistakes from giving too short a time to a convalescence after acute disease than too long, and the excuse of the expense or value of time has not always been present. Where there is a morbidness of character or a likelihood of a long rest developing into a state of permanent idleness, then a different mode of treatment is advisable. Here, again, a broad view must be taken of the whole surrounding circumstances, and action carried out in accordance therewith.

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A CLINICAL LECTURE

ON THE

EARLY SIGNS AND SYMPTOMS OF TABES AND GENERAL PARALYSIS.

Delivered at the Medical Graduates' College and Polyclinic, April 23rd, 1902,

By F. W. MOTT, M.D., F.R.C.P., F.R.S.

GENTLEMEN,—I have chosen this subject because it forms part, and perhaps the most practical part, of the work which I have been doing for the last few years. The two diseases, tabes and general paralysis, are, in my opinion, the same morbid process attacking different parts of the nervous system, and I base this statement upon clinical and pathological observations of eighty cases of tabes and sixty cases of the tabetic form of general paralysis. Etiologically the most important by far, if not the essential factor, is the syphilitic poison. Both diseases affect males more than females; both diseases occur on an average fifteen years after infection, though the limits vary between four and twenty-five years; both diseases are unknown where syphilis is unknown; both diseases occur especially in the fourth and fifth decades. A definite history of infection can be obtained in as large a number of cases as in syphilitic brain disease, or syphilitic skin disease. A considerable number of cases of married couples have been reported. In a number of these one may have suffered with tabes, another with general paralysis. Among my 140 cases, there were six of such. When either of these diseases affects young people, a history of congenital syphilis can nearly always be obtained. A number of cases, about 10 per cent., of tabes end in general paralysis, and about a similar number of general paralytics in asylums show typical cord lesions of tabes dorsalis. Both diseases are, in my opinion, a primary neuronic degeneration which may commence in almost any

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part of the central nervous system; but we may recognise the following types—spinal, medullary, optic, cerebral.

The name tabes indicates wasting, and when we look at either the brain of a typical general paralytic, the optic nerves of a blind tabetic, or the spinal cord of a case of locomotor ataxy, the one striking feature is the wasting.

Although there is a general similarity between all cases of spinal tabes and all cases of cerebral tabes (general paralysis), yet no two cases are alike, because no two individuals are alike in temperament, nutrition, or the conditions of life under which they have lived, the complex of which has been the exciting causes of the degeneration.

A patient suffering with tabes will come to seek relief for some one of the symptoms, and not because of the definite physical signs, such as the condition of the pupils, the knee-jerks, or Romberg's symptom. And it is well, therefore, to consider what are these early symptoms. They are such as to cause pain, discomfort, or interference with his business or distress to his friends, visceral disturbance, locomotor troubles. Such patients, if they go to the hospital, will be seen by the physician. A spontaneous dislocation or fracture will take him to the surgeon, and very possibly bladder trouble. A squint, with double vision or failing sight, ending, perhaps, rapidly in blindness, will take him to the ophthalmic department or ophthalmic hospital. A fit or mental symptoms will take him to the neurologist or alienist.

Each of these modes of onset of the disease is indicative of a special localised degeneration of some part of the nervous system.

I will then first consider these subjective symptoms, for they may first bring the patient under your notice.

Pains.—Shooting, darting pains in the legs, coming on in paroxysms, and being described as "lightning," are often thought to be rheumatic. When they occur in the thorax they are considered to be pleurodynia or pleurisy. If shooting down the arms, especially down the inner side, as they are apt to do, associated with sudden paroxysms of constriction of the chest, they may be thought to be anginal in origin, or due to pressure of an aneurysm. Pains I have found to be the most common early symptom, both in tabes and tabo-paralysis.

Visceral Disturbance.—A patient may seek relief for bladder trouble, even come with a distended bladder or he is unable to pass water without considerable exertion, or he suddenly has to run to pass water owing to the slightest cough or exertion causing an escape of urine into the urethra, which sets up the reflex process. The former is due to an atonic condition of the detrusor, the latter to an atonic condition of the sphincter.

Gastric Crises.—A patient may come for paroxysms of vomiting occurring even without food in the stomach, lasting for some hours, or even a day or more, attended with great pain, perhaps constriction round the trunk. This condition I have found to be relatively frequent, and may be present without absent knee-jerks or any sign of ataxy, the reason being that the diseased process has commenced in the spinal roots of the mid-dorsal region. There are, however, certain sensory disturbances of the skin of the trunk invariably present in these cases.

Rectal Crises.—A patient may come complaining of paroxysms of pain, of tenderness, and a feeling as if a hot iron were being passed up the rectum. I have known a case treated for piles.

Laryngeal Crises are not very frequently met with, but are an early symptom in some cases. Enquiry of Dr. Bond has surprised me that very few cases come to the Throat Hospital. Sudden attacks of adductor spasm of the laryngeal muscles with stridulous breathing and dyspnœa occur.

Locomotor Troubles.—A patient may come with early signs of locomotor ataxy; he may have had the lightning pains or one of the other symptoms in a mild form, especially bladder trouble, but these have not been of sufficient urgency to cause him to seek relief before a difficulty in walking occurred. He has noticed a swaying of his body in washing his face, or a difficulty in walking in the dark or going upstairs, or he may have had a sudden giving-way of the legs, as pointed out by Dr. Buzzard.

Optic Symptoms.—A number of cases find their way to the eye hospital for failing vision in one or both eyes. Sometimes they do not go there until blind in one eye, for when this occurs the inconvenience to their work urges them; it is then found that they have a limitation of the field of vision and optic atrophy. This is a very early condition indeed, and patients may remain a great

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many years in the pre-ataxic stage. In fact, some authorities give some comfort to their patients by telling them that they may remain a long period without further serious trouble. This is true. One of the patients I saw was twenty years before developing ataxy, and then he could not understand how he could have locomotor ataxy when he could walk ten miles. However, my experience shows that optic atrophy is very liable to be followed by other symptoms indicating brain degeneration. A very large proportion of the cases of tabo-paralysis in asylums are the subjects of optic atrophy, and frequently one found that the worry occasioned by their becoming blind had led to an attack of mania or melancholia, which had ended in progressive paralysis.

Double vision from a paralysis of an ocular muscle, usually in one eye, is a very common early symptom. The paralysis is usually transitory, but the double vision occasioned by a squint, which may not be perceived by the patient, is sufficient, on account of the inconvenience it causes him, to take him to the eye hospital. I had the opportunity thus of seeing a number of cases of very early tabes sent to me from the Westminster Ophthalmic Hospital to Charing Cross, also from Moorfields by my friend Mr. Treacher Collins.

Cerebral Symptoms.—Mania or melancholia may occur, or even delusional insanity, owing to insane interpretation of the symptoms, in a patient the subject of early tabes, and the mental symptoms may be the first which bring him under the observation of a physician.

We have next to consider the early symptoms of tabetic paralysis and general paralysis.

Seizures.—Sudden epileptiform, apoplectiform, or migrainous attacks, accompanied by temporary, partial, or complete loss of consciousness, followed often by some form of transitory aphasia, amnesia, word-blindness, or word-deafness lasting a few hours, a few days, or a week or two, may be the first indication of the disease. If the attacks are slight the patient or his friends may neglect to seek advice, indeed, these attacks may be so slight as to occasion only a slight lapse of consciousness, which the wife may tell you was "a look of bewilderment." These slight attacks may precede a severe attack, so severe, indeed, that it may be considered to be an attack of apoplexy from which the patient will not recover. A few days later he is sitting up, and within a week, perhaps, apparently quite himself. I say apparently because very frequently, on close questioning the wife, many prodromal indications of the serious and insidious disease, which will, in a few years, prove fatal, have been observed by her. And all cases of general paralysis probably show this prodromal stage if they are carefully investigated.

Mental Symptoms.—The psychical condition of an individual, especially regarding his emotions and feelings, continually oscillates around a mean point of equilibrium. The extent of the oscillations within the limits of health depend upon bodily condition, temperament, and environment. Above and below the mean point of equilibrium are grades of pleasure and of pain, which are reflected to the muscles of expression, causing synergic alterations in their minute tensions. Every passing feeling is thus reflected, so that there is no art "to find the mind's construction in the face," but it is only the wife or closest associates who can guage this alteration in the feelings and emotions and their reflections in facial expression. They alone have known the man as he was and as he is; still, the expert who has had a large experience of this disease may almost intuitively, from a study of the facial expression, so suspect its existence as to make further enquiries and look for physical signs.

However difficult as it is to fix the normal in the physical organism, how much more so is it in the psychical? Only those who have had the opportunity of observing the feelings and emotions, the intellect and actions of the individual closely and for some considerable time can correctly appreciate a change of character, volition, and intelligence. Sleeplessness and nervousness are the symptoms which may bring him to the doctor in this stage.

Long before the patient has shown the more obvious and obtrusive symptoms and signs of the disease, the wife or partner in business or some close associate has noticed a change of character. Previously affectionate, kind, of an equable temperament and happy disposition, he has become subject to fits of temper, is sullen, morose, or depressed, often neglectful of his wife and children, or he worries over the least trifle; accustomed to act independently, confident of his own judgment, he loses all self-reliance, and turns to others for advice in the smallest matters. He may be restless and excitable, or sullen or moping, and he

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has become a man of varied moods, unable to fix his attention on any subject for any period of time, or the history sometimes points to the patient having been filled with morbid suspicions, which later become fixed delusions of persecution. The delusions or fits of temper may even impel him to attempt suicide or homicide, and the wife may be afraid of her life and that of her children. Other instances occur of quiet and sedate, even staid individuals exhibiting an antithesis of character, giving way to amusement, gambling, immorality, vice, and intemperance. Sometimes the prodromal period may be marked by flashes of genius or exceptional brilliancy of artistic or intellectual power. There is always, however, a failure of concentration and steadfastness of purpose to carry the schemes to fruition. The patient may become careless about his personal appearance, untidy in his dress, forgetful of his business or his home affairs, especially in regard to events that have recently happened, his mind being absorbed by ambitious schemes which come to naught. Signs such as these may end in an attack of mania or melancholia.

Physical Signs.—There is one which, if in conjunction with one or more of the subjective symptoms above enumerated, is almost sufficient upon which to make a diagnosis, and that is the Argyll-Robertson pupils. Unequal pupils also are very suggestive. In 80 per cent. of all cases of tabes, the Argyll-Robertson pupil is present, and in general paralysis it is present in about half the cases, but sluggish reaction to light and activity to accommodation generally occurs in the remaining cases.

The prodromal mental symptoms which I have enumerated in the absence of pupil phenomena, even with a history of syphilis, would not justify a certain diagnosis of general paralysis, for all these symptoms might occur in neurasthenia.

The Knee-jerks are very early abolished in the majority of cases of tabes, but a number of cases do occur with optic atrophy or ocular paralysis, or gastric and laryngeal crises in which the knee-jerk may be present, and even exaggerated; as, too, in cases of arm tabes.

In some cases the knee-jerks have disappeared, first on one side and then on the other, after attacks of pains in the legs. It may be present on one side, and absent on the other. This may be due to the fact that the roots concerned with the sensory innervation of the quadriceps extensor muscle have not yet been destroyed on one side; or it may be due to the fact that there is coincidently a progressive degenerative change occurring in the opposite hemisphere. Such cases portend badly, for they indicate pyramidal degeneration due to changes of a similar nature to general paralysis in the cerebral cortex. In general paralysis, as a rule, the knee-jerks are exaggerated, except in the tabetic form, where there is simultaneously with the brain degeneration, spinal cord degeneration.

Disturbances of Cutaneous Sensibility should be sought for, as they are among the earliest objective phenomena of the disease; particularly have I found anæsthesia or diminished sensibility to light tactile sensations in the thoracic region corresponding to the fourth, fifth, and sixth dorsal segments, and extending above and below this to other segments.

DEBATE

ON

CANCER: ITS ORIGIN, NATURE, AND GENERAL PRINCIPLES OF TREATMENT.

Held under the auspices of the Chelsea Clinical Society, at the Jenner Institute of Preventive Medicine, March 11th, 18th, and 25th, 1902.

Dr. CHARLES MORRIS, C.V.O., President, in the Chair.

The Chairman.—As you all know, the subject of our debate is "Cancer: its Origin, Nature, and General Principles of Treatment." This is the fourth annual debate which we have held, and our custom is for the Council to choose a subject which they think will be generally interesting. I think you will agree that the subject which we have chosen for this year is one which is of general interest. It appeals to us, as members of the medical profession, and judging by the amount which is written about it in the daily press, it is evidently interesting to the general public too. You all remember, I dare say, when the foreign delegates in connection with the Tuberculosis Con-

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gress were received by the King, His Majesty said that the cure of cancer was the subject which should occupy the attention of medical men, and that the man who found a cure for the disease deserved to have a monument put up in every city in the world. I imagine that probably this disease excites more interest in the minds of the public than does any other. It has a sort of horror hanging over it; it seems invariably fatal to its victims, and we know of no cure. Of course our subject does not in any way lead us to expect that any of those who will speak to-night will tell us that they have found the cure of cancer, or that they have even discovered the cause of the disease. Our purpose in having this debate is, that so far as possible, we shall be up to date in the knowledge of the present day on subject of cancer. Perhaps the day is not very far distant when the cause and cure of cancer may be discovered, but we can hardly expect it to-night. I think the papers and speeches will be both varied and interesting, and I will mention those who we hope will take part in the debate. We have had to make a few alterations in our original programme. Our first intention was that bacteriology should form the chief part of the debate, but on asking the bacteriologists we found that many were very loth to in any way commit themselves to definite statements on the bacteriology of the disease. I think that most of us here to-night are inclined to look upon cancer as a disease due to parasites, but there are very strong opponents of that view. This evening, or at any rate during our debate, I believe we shall hear those who advance both views, namely, those who think that it is bacterial in origin, and those who do not. I hope we shall be in a position at the end of the debate to form a definite opinion upon the subject. As we found some bacteriologists did not care to commit themselves, and we could not have a full debate from bacteriologists only, we have enlarged the subject, and made it to include the general principles of treatment. You will see from the list of the speakers this has greatly increased the interest of the subject. To-night Dr. Galloway will open the subject, Dr. Tatham will follow, then Dr. Beatson and Mr. D'Arcy Power. We had so much material left for the second night that we found it would be impossible to get it finished in the time at our disposal, and we accordingly thought it advisable to make the debate extend to three evenings instead of two. Next week Sir Mitchell Banks will open the meeting, Dr. Blacker will give his demonstration of the treatment by X rays and ultra-violet rays, and Mr. Pearce Gould, Mr. Mayo Robson, and Mr. Sheild—either all or some,—will take part. On the third evening Professor Hewlett and Dr. Payne will be here, and, if they arrive, the papers which are being sent by Dr. Gaylord and Professor Adami will be read.

Dr. GALLOWAY.—Mr. President and gentlemen, I am sure no one feels more keenly than I do at the present time that Dr. Payne is not present to open the debate this evening, because in the present condition of our knowledge as to the origin of cancer I am sure that his rich clinical and pathological experience would be of the greatest value to us all in helping us to form some sort of conception of the present state of our knowledge, and I am glad to hear he will be present at a subsequent meeting, and that we shall hear his views on this question. This is especially the case when I hear of certain pathologists who consider that the present moment is a very inopportune one for discussing the question of the origin of cancer. So much has been written in previous years on the subject, and so much that has been written has been found to be not quite sound, that naturally enough those who are engaged in observations at the present moment feel disinclined to commit themselves. But it appears to me that this is probably a proper time for considering the present state of our knowledge, and for noting especially the alterations which have taken place in the various standpoints which are occupied by pathologists owing to the observations of the last eight or ten years. Eight or ten years ago, when observations were made on which were founded parasitic theories of various forms of cancer, a great deal of hope arose in the minds of the medical profession that at length some sort of explanation could possibly be given as to the origin of the disease. Although many of these observations, I think, were not well founded, and at the present time cannot be said to have withstood the criticism of experience, still, the work which was done at that time was of great value, and, at any rate, showed us the way in which future observations should be carried out. And, even in the matter of treatment—which is the most

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important of all the considerations which we have to have in our minds—I think we may say, as the result of observations and the stimulus which was given to research by the statement of the parasitic hypothesis of the cause of cancer, the ordinary surgical treatment of cancer was greatly changed, and made much more effective. We learned the rapid and extensive way in which the growth spreads, and the operations which came into vogue shortly after that period were very much more complete than those which had ever been carried out before. And the observations have led to experiments in new methods of treatment, upon some of which Dr. Beatson may feel inclined to enlighten us this evening, which may bear still further fruit. Although many of the observations which were made at that time were apparently fruitless from the point of view of the origin of the disease, they have given rise to very considerable benefit in many ways.

I suppose none of the actual bodies and substances or materials which were discovered in cancer at the time of the investigations to which I have alluded, and described as of parasitic origin, have withstood the test of criticism. Some of these I shall refer to afterwards, and, if the Chairman wishes and there is time, we will see some slides which I have brought, which I think may be of interest to the meeting. But the other theories have also done very good service, I recollect very well when the parasitic theories were so much supported, and when the earlier and very important observations of Cohnheim as to the origin of malignant growths from residual remnants of embryonic structure were discussed, that this theory was not exactly scouted, because the name which supported it claimed too much respect. Still, very little consideration was given to it. But even a hypothesis of this nature, which was found very largely not to account for the facts, has helped us a great deal, and we have always understood that some forms of growth, at any rate, perhaps more of the nature of deformities than tumours, were actually due to embryonic remnants. And more recent observations, especially on the forms of growth which arise in nævoid tumours, have shown that the Cohnheim theory is perhaps of a little wider application than was thought at that period. Every one knows, from clinical experience, that forms of malignant growth, sometimes very virulent indeed, arise from various forms of nævi. These growths for a long time were considered to be of sarcomatous character. Later on, on account of the nature of the cells, they were considered to be endotheliomatous, that is to say, composed of peculiar mesoblastic cells, which resemble endothelial cells in some respects, but behave as epiblastic cells in other ways. Observation and experiment on this character of growth was stimulated, and we know that many of these tumours arising in nævi and these embryonic remnants really take origin from cells which are derived from epiblast which has been left in the process of development, and at some later period take on tumour formation. Still, the point remains that although we can account for epithelial growth in this way, the great secret of the malignancy of these growths yet remains unexplained, even in such cases as this. The point to remember in reference to the malignancy of the growth is not simply the power which the epithelial cells have to multiply, but they acquire a new attribute: they are capable of passing from the position in which they originally were arranged to positions in which they ought not to exist, and they are capable of multiplying in these new situations, and probably of undergoing still further changes, of which we know very little. A certain amount of new information, therefore, has been acquired, even in reference to the older hypotheses.

As a result, also, of observations, due to the stimulus of the parasitic hypothesis, we know now more about certain forms of epithelial overgrowth; those, for instance, which are recognised by ophthalmic surgeons as implantation cysts; also those which are recognised by surgeons and by dermatologists as epithelial masses existing deep in the cutis, quite apart from the epidermis, and due to, or arising at the site of an injury. It appears in both these cases that living tissues carried deeply into the cutis, on the one hand, and into the mesoblastic tissues on the other hand, are capable of reproduction and development, and tumours form in these situations. Still, we are confronted with the difficulty that we cannot explain in this way why, under certain circumstances, these masses of epithelial tissue or growth should take on malignant characters. Why is it that they assume the power of migrating from the situation in which they have developed and grown, and pass

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rapidly along the lymphatics and other channels to positions in which they ought never to exist?

Passing now to the consideration of the parasitic hypothesis, as I have just remarked, I do not know that any one would feel inclined, even the most enthusiastic supporters of the parasitic hypothesis, to say the various "cancer bodies" which were described at that time are really of a parasitic nature. I know a recent paper by Dr. Gaylord states that he was inclined to accept as parasitic structures many that we had considered as proved definitely not to be parasitic, but when his paper, now in the "Etruria," comes to hand, I hope we shall have further information on this point. Although at the time I alluded to I was very much attracted to the hypothesis, and tried to believe that some of these things might be of a parasitic nature, I found that the ground became much less firm as I went on to investigate. At one time it was hoped that much information could be derived from the study of certain epithelial overgrowths which occurred in men, and also in the lower animals, which were undoubtedly of a parasitic nature, and in which protozoa unquestionably did fulfil a true causative *rôle*. Numerous tumours of this sort occur, and the one which was most investigated, and of which one heard of most on account of its ease of investigation, was the class of peculiar parasitic tumours which occur in rabbits, due to a well-known protozoon, the *Coccidium oviforme*. But a very important point arose out of this investigation, namely, that these protozoa are very easily recognised; even on an ordinary microscopical examination there is no question whatever of difficulty in the recognition of them. Still more, the process of reproduction and multiplication was very readily seen in these small animals, and that is a state of affairs which is entirely different from the hypothetical parasitic bodies which were described in the various forms of cancer. I think no process which was supposed to be representative of reproduction in these so-called cancer bodies has withstood criticism. On the other hand, as I have remarked, not only are these protozoal structures in the rabbits capable of easy recognition, but there is very little difficulty in seeing the process of multiplication, not only in the rabbit itself, but outside the host; in the latter condition any one who takes the trouble to make a cultivation can watch them for himself. Recognising that perhaps some members of this Society may not have seen these actual protozoal organisms, I took the liberty of bringing with me a few lantern slides to demonstrate the position of the organism in the host, and also the changes which can be brought about by cultivating the coccidium in an ordinary temperature in a moist chamber without any special arrangement of heating—in your own consulting studies. I will just run these slides through the lantern to revive your recollection of the structures in question. The disease is a very common one in rabbits, and sometimes produces such epidemics as practically to kill out whole broods on account of the changes which are produced. The first slide is a low-power view of a section of one of the nodules which the parasite causes in the bile-ducts of the rabbit. You will observe the state of affairs. This is the nodule, and the normal liver tissue, and also a section of the portal vein. You see a distinct tumour is produced. There are certain dissepiments, which are due to the bifurcations of the bile-ducts. You will notice that great proliferation of epithelium has occurred; the firm processes which project are very elaborate convolutions of the epithelial lining of the bile-duct. But I wish you to observe that in this case the epithelium, which is, of course, enormously increased in amount, retains its original position. A well-marked basement membrane exists, and the liver tissue is not invaded, except by the pressure of the mass. There is no place at which the epithelium is undergoing changes, so that its cells pass into the liver tissue. Next I show you a specimen, much more highly magnified, of a tumour such as we have just viewed. You will observe here, again, the convolutions which I have mentioned and the lining membrane of the bile-duct. They are very elaborate, producing fine branchings, and each of them lined by epithelium; and in the epithelium, if you look carefully, you will notice numbers of bodies. These are encysted protozoa, and they are very numerous indeed. Here is another section from a similar tumour, and you will be able to notice the large numbers of parasites which exist. I think it may be fairly asked, even in this case, whether this great multiplication of epithelium is not due rather to the blocking up of the bile-duct by parasites and to irritants which the parasites may produce, and is really an overgrowth of adenomatous nature,

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due to irritation rather than to the parasites themselves. I think it is doubtful that the reproduction of epithelium is due to the parasites, though perhaps the parasites may have a certain effect. The next slide shows the final stage in one of these nodules. Gradually the epithelium degenerates and disappears, and finally the little tumour contains the capsules of the parasite in large number, and very probably the animals themselves actually die. It is very difficult to get cultures from the growth in this stage. I have brought one or two slides to show you how easy it is to see the processes of reproduction and multiplication in this organism. This represents the cycle of development outside the host. The cultures were made simply by taking a little scraping from one of the nodules, placing it upon a microscope slip in a moist chamber, and keeping it in my own study. You will observe the peculiar cysts surrounding the parasites in the resting condition; they are not like any ordinary living human cell. This photograph was taken twenty-four hours after the cultivation commenced. These are the living animals, and that fact accounts for the dirty appearance of the lantern slides; they were not specially cleaned and prepared. I have here two diagrams which will serve to interpret those which are coming later. They do not represent very accurately the state of affairs, but you will see that the single ball of protoplasm divides into four. This next picture represents the reproduction of a coccidium closely allied to the *Coccidium oviforme*. There is no difficulty in seeing the rapid division of the ball of protoplasm, which represents the organism in the rest state. Here I show you the second stage of cultivation. It is one of those capsules which you have just seen, in which the ball of protoplasm has divided distinctly into four. Here is a cultivation in which division has taken place very profusely indeed. There are numerous capsules. Here is one magnified to a very much higher power, and even in this preparation I think you will see that four distinct balls have appeared instead of one, which was the characteristic of the parasite in the early stage. So there is no difficulty about recognising the fact that when you have protozoa of this type they do divide. It is easily seen. Another process of division takes place within the host itself, in the intestine especially; it is a little different in this type, but it can also, with some difficulty, be made out. So the difficulties of accepting the protozoal parasitic origin of the various forms of malignant growth became very great.

I notice at the present time the tendency amongst observers is to consider that some form of fungoid growth may be the actual cause of the formation of malignant tumours. This is a very difficult hypothesis indeed to accept, and it is difficult first of all for this reason: that fungi, of apparently the same nature as nearly as possible,—one might say exactly the same nature—have been described by pathologists as producing tumours of a totally different character. Pathologists are familiar with the descriptions which have been given of blastomycosis and blastomycetic dermatitis, a condition of the skin and other tissues in which yeast-like fungi have been first of all isolated, and apparently after cultivation have produced similar growths on inoculation. The difficulty which strikes one at once is this: that it is very difficult for us to accept the fact that a budding fungus should be capable of producing a tumour such as that of blastomycosis, which is really a granuloma resembling very much in structure the granulomatous tissue in tuberculosis. It is difficult for us to conceive that it should be capable of producing a tumour of this nature, and also the very much more elaborate structure which one sees in carcinoma and sarcoma. And there is a second difficulty—which one mentions with very considerable hesitation, because the observations are very few,—arising from the fact that apparently tumours, and malignant growths also, can be infected accidentally by fungi-producing spores, and even fungi of a little higher development, producing hyphæ. I have a section under this microscope on which I should like to have the opinion of certain gentlemen whom I see present this evening, from a case of carcinoma of the skin. The carcinoma was sent in the ordinary way for the purposes of diagnosis. The diagnosis was very easily made; it is columnar carcinoma of very ordinary type. But when it came to be investigated more closely—I forget for what reason—a second time, it was found that certain parts of the tumour, certain degenerating portions of it especially, contained large quantities of a fungus-producing hyphæ—threads—with distinct segmentation and spores. The portions of the tumour which were so infected

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were deep, and were not related to the surface. This particular tumour was not obviously ulcerated at the time it was excised, and yet deep in that tumour were the mycelium and hyphæ of a fungus, and, of course, spores at the same time. Now, supposing a culture had been made of a superficial portion of the tumour, near one of these seats of degeneration, it would have been easy to obtain some form of culture. These fungi are not difficult to grow, and a cultivation would have been obtained of fungus-producing spores, possibly simply a budding fungus, and a very suggestive diagnosis might have been made. So we have difficulties in the way of accepting the hypothesis that the budding fungi are related causally to carcinoma, in the first instance because these budding fungi are claimed to produce tumours of the nature of granulomata; and we have a shrewd suspicion that growths may be infected quite accidentally by fungi of the same nature.

It is from our clinical experience that we are all likely to inquire as to the truth or falseness of a pathological theory, and it must be admitted that clinical evidence gives a very grudging and scant support to the parasitic theory of the origin of cancer. There is an interesting body of clinical evidence, which has not been much appealed to as yet in considering this question, derived from those diseases in which prodromal morbid states precede the development of cancer. For instance, in Paget's disease of the nipple we have a prodromal state, but in that, according to the evidence of some observers, the prodromal state is too nearly allied to cancer itself to present a good example. These authors would call that disease a malignant papillary dermatitis. Some of you may have noted observations which appeared in American papers in which the analogies between the multiplication of cells in Paget's disease and epithelioma are shown to be so close that one would feel disinclined to consider Paget's disease as prodromal in the sense to which I now allude. I refer particularly to such conditions as the rare disease xerodermia pigmentosa, and certain forms of abnormal pigmentation. Xerodermia pigmentosa, it will be in the recollection of most of us, is a disease which occurs in family groups, preceded by a deposition of pigmentation, and leading to the development of epitheliomatous tumours which prove fatal. In this abnormal condition there occurs, possibly for years before the development of carcinoma which proves fatal, an alteration in the texture of the integument. In the former — xerodermia — the change is characterised by the development of pigmentation resembling exaggerated freckles, accompanied by atrophy of the skin, and degeneration takes place. In the latter melanin is deposited about the clefts of the fingers and toes and nails, in localised areas, with clearly marked margins. In such cases it is only a question of time before, first of all, breaking down of the surface occurs, and finally the development of true epithelioma comes about, usually melanotic. It is surely more easy to conceive of these changes as being successive manifestations of a progressive process of degeneration, the earlier signs being alteration of the pigment distribution, and the later the disorganisation of the means by which the growth of the epithelium is kept in check and properly distributed and arranged. And if it is conceded that the distribution of pigment is very directly influenced by the nervous system, may not the process continue, and the disorganisation of nervous control end in allowing the resistance of the connective tissues to be impaired, and the epithelium to escape from control? So also in the prodromal conditions, of which senile keratoma is an example, from which arise epithelial cancers of varying degrees of malignancy; is it not an easier conception to hold that the whole course of the disease is continuous than to invoke the action of a parasite, when at last the epithelial cells are so affected as to show evidence of the peculiar form of overgrowth which we recognise as "malignant"?

Considerations such as these lead to discussions on the subject of the "physiological equilibrium" of the tissues and its alteration in disease, so interestingly placed before us by Dr. C. P. White in his Erasmus Wilson lectures, but which, at the present time, are unfortunately very fruitless because we do not know the factors which maintain and interfere with this condition. It must be the desire of every one that observation and experiment will, in the near future, tell us why the balance is upset, and why a tissue-cell, instead of leading its normal life, suddenly runs riot, penetrates beyond bounds, and multiplies with an energy never approached in its usual normal, well-ordered existence.

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Dr. J. F. W. TATHAM, Superintendent of Statistics, General Register Office.—Mr. President and gentlemen, I cannot contribute anything to this discussion from the side of pathology, or from the side of clinical medicine. I need hardly explain to you why that is the case. I am very glad, however, to be able to present to you a statement, I hope fairly accurate, of the mischief which cancer—by which I mean malignant disease in its broadest sense—is doing, and has been doing for many years past. In England and Wales cancer is fatal to something like 26,000 people a year. That is a terrible total, and I am sure that in view of whatever investigation you may make into the causes of cancer, a very good preliminary will be an accurate statement of the actual mortality for which this disease is responsible. I will accordingly ask you to allow me to read the few remarks which I have prepared bearing upon this point.

The mortality from cancer is not very excessive until some time after the twenty-fifth year of age. Thus at ages under thirty-five it averages from 44 to 66 per million, but as age advances the mortality increases in both sexes very rapidly, until at the more advanced ages it is enormous. The following table shows this:

Annual Death-rate per million living at Each Age.

	Males.	Females.
All ages . .	672	975
Under 35 . .	44	66
„ 35—45 .	418	942
„ 45—55 .	1483	2433
„ 55—65 .	3796	4561
„ 65—75 .	5735	6254
Above 75 . .	6715	7468

In England and Wales during the last four years the deaths from malignant disease have averaged more than 25,000 a year. In the returns issued from the General Register Office, the term "malignant disease" includes all the diseases classed as such in the Nomenclature of the Royal College of Physicians of London. Hitherto all forms of malignant disease have been grouped together under one heading in our national returns, but with the new century a revised list of diseases has been adopted, of which a shortened form is published for London in the weekly return. In future issues of the official book of forms of death certificate, a copy of the new list of diseases in full will be inserted, together with suggestions for the assistance of medical practitioners in filling up these certificates. In this list the carcinomata, or true cancers, are distinguished from the sarcomata, and these, again, are distinguished from the indefinite forms of disease which are ascribed in medical certificates to "malignant disease," or "cancer" merely, without further qualification. That medical practitioners appreciate this improvement in the classification of a most important group of maladies is shown by the fact that in London, since the beginning of the present year, when the new list of diseases was first published, the 800 aggregate deaths from malignant disease have been differentiated thus: in the medical certificates supplied for registration 52 per cent. have been certified as from carcinoma, 6 per cent. from sarcoma, and the remainder, amounting to considerably less than half, to "cancer" or malignant disease. It is highly gratifying to me, as I am sure it will be to you, gentlemen, to note how readily the practising members of our profession respond to any attempt to secure greater accuracy in the statistical records of the fatal cases coming under their care. And I would take the present opportunity of cordially thanking many of the physicians and surgeons here present for the courtesy and kindness with which they have invariably answered my requests for information respecting their deceased patients further than that which can be got into the ordinary form of certificate.

Malignant disease in the aggregate, *i. e.* without limitation as to organ or part affected, is more fatal to women than to men.

According to the latest returns, the male death-rate from cancer in England and Wales was equal to 672 in each million living, while the female death-rate amounted to not less than 975 per million. But this excess is due to the well-known tendency of malignant disease to attack the generative organs of the female rather than those of the male.

On reference to the following table, it will be seen that when the deaths from cancerous affections of the ovaries, uterus, and breast are eliminated from the total cancer deaths of females, the remainder give a death-rate among females

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which is considerably below that among males. Thus, in the four years 1897 to 1900, the male deaths from cancer, less the deaths from disease of the organs referred to, correspond to a death-rate of 645 per million, whilst the female rate, with the same limitations, did not exceed 568 per million.

Cancer Mortality, 1897—1900 (*total deaths in the four years*, 102,685).

Parts affected.	Deaths.		Rate per million living.	
	Male.	Female.	Male.	Female.
All parts	40317	62368	657	952
Lip	647	74	11	1
Mouth	508	115	8	2
Tongue	2124	271	35	4
Throat	891	334	15	5
Œsophagus	2358	852	38	13
Stomach	8369	8355	136	127
Intestines	6312	6731	103	103
Liver	5532	8654	90	132
Generative and mammary organs	726	25151	12	384

It is notorious that in recent years the mortality from cancer has increased very rapidly, not only among females but among males. This is shown by the subjoined figures:

Annual Mortality in England and Wales. Rate per million living.

Males.		Females.		Increase in 1891–1900 per cent.	
1861–70.	1891–1900.	1861–70.	1891–1900.	Males.	Females.
242	597	519	903	147	74

It thus appears that although at the present time women suffer much more severely than men from malignant disease, nevertheless the disease during the last thirty years has increased among men much more rapidly than among women.

Dr. GALLOWAY then showed a further collection of slides, and demonstrated them. He said: I would remind you that these slides are simply to revive one's recollections rather than to show anything new. These are sections of carcinoma that show various bodies which have been described as protozoal parasites. These are actual sections of the tissue, and they are magnified to a considerable extent, which may account for the difficulty of definition. This is a portion of the acinus of a carcinoma of the breast, in which the pavement-like arrangement of the cuboidal cells can be seen. Here is a cell in which a body of a different nature from an ordinary nucleus can be seen. This was the structure, above all others, which was studied by pathologists from the point of view of its being possibly a parasitic organism, a protozoon, and causing by its presence a malignant multiplication of cells. You will notice the cancerous cell and a very well marked distinct body. This structure, I think, can be explained by the fact that it is a peculiar degeneration of the protoplasm itself. The next slide shows a section representing the same sort of appearance, a rapidly growing carcinoma undergoing rapid division, and in the lower portion you will see the cell where there is an equatorial plate, one of the stages of karyokinetic division. I show you several slides of the same sort. You will notice in these structures a dark spot in the centre which looks like a nucleus; these stain in various ways, as was found by Ehrlich and others. We had not met with them distinctly before the new methods of staining were discovered, and they were quite new to pathologists and histologists at the time. The tendency was to consider them as being something quite different from other structures, and therefore possibly parasites. Sometimes one can notice attempts at division. Sometimes these peculiar looking bodies appear in great numbers, and I do not know that one can say the actual rapidity and malignancy of the growth has anything to do with the number of these cell bodies present. Here is one magnified much more highly. Here is a large body occupying the free protoplasm of the cell, with a peculiar organisation which seems to suggest an actual vital structure. Here is another form, which is probably of a different sort, an invaginated cell which is put into a larger cell, and the two have been cut across. I think in all cases these cancer bodies have been sufficiently accounted for by the process of inclusion of one cell in another, or as being due to the degeneration of protoplasm or nuclear material in various ways.

(*To be continued.*)

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HYPNOTISM:

AN OUTLINE SKETCH.

Being a Lecture delivered before the King's College Medical Society.

By J. MILNE BRAMWELL, M.B., C.M.

(*Concluded from p.* 45.)

I FIRST went to Nancy to attend Liébeault's clinique, but before telling you what I saw there I wish to give you a slight sketch of Liébeault and his career. He was born in 1823, and was first attracted by mesmerism during his student days, when he made some successful experiments, but it was not until 1860, after many years of general practice, that he renewed his researches. He found he could frequently induce a condition by suggestion, which he regarded as analogous to sleep, and termed *sommeil provoqué*.

In order to find subjects for experiment, Liébeault took advantage of the parsimonious character of the French peasant. His patients had absolute confidence in him, but they had been accustomed to be treated in the ordinary manner. He therefore said to them, "If you want drugs I will give you them, but you will have to pay me as usual. On the other hand, if you will allow me to hypnotise you I will do it for nothing." He soon had so many patients that he was unable to find time for necessary rest or study. In 1864 he gave up general practice and settled in Nancy. He had managed to save enough to live quietly, and devoted himself to hypnotic research and to the gratuitous practise of hypnotism amongst the poor. For two years he worked hard at his book, 'Du Sommeil, et des États analogues,' but of this one copy alone was sold. His colleagues regarded him as a madman, the poor as their providence, calling him "the good father Liébeault."

In 1882 a patient who had suffered from sciatica for six years entered Bernheim's wards. Six months later he was discharged unrelieved, and consulted Liébeault, who quickly cured him. Hearing of this Bernheim visited Liébeault's clinique. At first he was sceptical and incredulous as to what he saw, but soon his interest was excited; he multiplied his visits and became an eager pupil. In 1884 Bernheim published the first part of his book 'De la Suggestion,' completed in June, 1886, by 'La Thérapeutique suggestive.' From this date, Liébeault's name became widely known, the first edition of his book was quickly bought up, and doctors flocked from all countries to study the new therapeutic methods.

Presently, I will draw your attention to the fact that Braid not only anticipated the most important observations of the school of Nancy, but also escaped from some of the errors it fell into. We ought not, however, on that account to undervalue the services of that school, and more especially those of its founder Liébeault. Although Braid's researches excited interest in France, little or nothing was known of his works except 'Neurypnology,' and his last MS., which contained some of his later views, was not published in France until 1883. Liébeault independently arrived at the conclusion that the phenomena of mesmerism were purely subjective in their origin, and to him we owe the development of modern hypnotism.

Another point in reference to their career is worthy of note. Braid's views at once brought him fame. His books sold rapidly, the demand for them exceeding his power of supply. The medical journals were open to him to an extent which may well excite envy in those interested in the subject at the present day. Liébeault's book, on the contrary, remained unsold, his statements only found sceptics, his methods of treatment were rejected without examination, and he was laughed at and despised by all. From the day he settled in Nancy, in 1864, until Bernheim—some twenty years later—was the means of bringing him into notice, Liébeault devoted himself entirely to the poor, and refused to accept a fee lest he should be regarded as attempting to make money by unrecognised methods. Even in his later days fortune never came to him, nor did he seek it, and his services—services which he himself, with true modesty, described as the contribution of a single brick to the edifice many were trying to build—only began to be appreciated when old age compelled him to retire from active work. Though his researches have been recognised, it is certain that they have not been estimated at their true value, and that members of a younger generation have reaped the reward which his devotion of a lifetime failed to obtain.

I found Liébeault's clinique invariably thronged, but there was neither excitement nor air of mystery, and everything was conducted in simple

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homely fashion. The patients chatted freely amongst themselves, and questioned the doctor in a more familiar way than one had been accustomed to see in England. Each in turn was placed in an arm-chair, told to think of nothing, and to look steadily at the operator. The fixation was not maintained long enough to produce any fatigue of the eyes, and appeared to be simply an artifice for arresting attention. If the eyes did not close spontaneously, Liébeault requested the patient to shut them, and then proceeded to make the following suggestions, or others resembling them :— "Your eyelids are getting heavy, your limbs feel numb, you are becoming more and more drowsy, etc." A certain proportion of the patients apparently passed into a deep hypnotic trance; others only became restful or drowsy. In each instance, curative suggestions suited to the individual case were repeated for about ten minutes. The patients were then told to awake, when they either walked quietly away or sat for a little to chat with their friends. Liébeault took especial pains to explain that he neither exercised nor possessed any mysterious power, and that all he did was simple and capable of scientific explanation. No drugs were given.

When I returned to Nancy in 1892, failing health had compelled Liébeault to abandon his clinique. He still, however, did a certain amount of hypnotic work, and had then under his care an interesting case of hereditary suicidal mania. This patient recovered under hypnotic treatment, and two years later Liébeault informed me that she still remained well.

At this time I also visited the Nancy Hospital, where Bernheim had hypnotised thousands of patients, the induction of hypnosis in his wards having become as much a matter of routine as taking the temperature or the pulse.

Amongst other foreign cliniques I have been to, there are three to which I wish specially to refer, namely, that carried on conjointly by van Eeden and van Renterghem, and those of Forel and Wetterstrand.

Forel, who till recently was Medical Director of the Burghölzli Asylum, and one of the professors at the University of Zürich, is well known, not only as a distinguished alienist, but also for his important entomological writings. After visiting Liébeault, Forel commenced to investigate the subject on his own account, and soon satisfied himself of its therapeutic and scientific interest, the origin and spread of the hypnotic movement in Germany being largely due to him. Before leaving Zürich, Forel had hypnotised over a thousand persons, many of whom were insane, and influenced about 95 per cent. In cases of undoubted insanity his results were disappointing, but he cites a long list of other diseases, in which he found hypnotic treatment markedly beneficial. Amongst his successful cases were several patients who suffered from confirmed dipsomania, and who had long been inmates of his asylum.

Forel also succeeded in hypnotising nearly all his asylum attendants, both male and female, a large proportion of them becoming profound somnambules.

In the 'Zeitschrift für Hypnotismus' for 1898, Dr. Inhelder, formerly assistant physician to Dr. Forel, records in detail the experiments they made for ten years (1887–97) in the night watching of dangerous lunatics. Hypnotised warders were trained to sleep by the bedside of these patients, and to awake the instant they heard them attempt to get out of bed, while sounds which had no import with reference to their duties were inhibited by suggestion. These warders sometimes performed night duty for six months, and worked hard all day without showing signs of fatigue. The results were uniformly successful, and no accident of any kind occurred.

In many instances Forel successfully excited and arrested menstruation by hypnotic and post-hypnotic suggestion; the female warders were the subjects of most of these experiments, which Forel claimed were conducted in such a rigorously scientific manner as to exclude all possible sources of error.

When I visited Forel in 1892 he showed me a number of patients undergoing hypnotic treatment, and gave me an interesting demonstration of the way he trained warders to watch dangerous patients. Forel has now given up asylum work in order to devote himself more fully to hypnotism, which he still employs with excellent results.

Amongst those who practise hypnotism few have been so successful as Dr. Otto Wetterstrand, of Stockholm. I am unable to quote his complete statistics, but up to January, 1893, he had attempted to hypnotise 6500 persons, of whom only

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3 per cent. were uninfluenced. The therapeutic results he claimed were so startling, especially in the treatment of epilepsy, that Forel stated it was only after personal investigation that he was able to accept them.

In August, 1894, I attended Wetterstrand's clinique for a fortnight, and, like Forel, was much impressed by his methods and therapeutic results. Amongst other things, he pointed out that owing to Liébeault's influence too much importance was attached to treatment by suggestion, and not enough to the curative effects of prolonged hypnotic sleep. He treated epilepsy and grave forms of nervous disorder by keeping the patients in the hypnotic trance for three or four weeks. During this time it was suggested that they should sleep deeply and be insensible to all communications from the outer world, except those coming from the operator, and the nurse who was put *en rapport* with them. Without rousing them, the patients were fed at stated intervals, and the action of the bowels and bladder regulated by suggestion. In this way a mental as well as physical rest cure was given, with the addition of the therapeutic advantages of suggestion.

Before visiting Sweden I had read Wetterstrand's account of this method, and had attempted to employ it in the case of a young girl suffering from epilepsy, whom I had previously hypnotised. Through the courtesy of a medical friend I was allowed to treat the patient in the New Hospital for Women; the experiment was a failure, and I went to see Wetterstrand to find out the cause. He told me it was necessary to visit the patient several times a day, and to repeat and reinforce the suggestion of continued sleep, otherwise hypnosis would terminate spontaneously, and the treatment be thus interrupted. I had not even paid my patient a daily visit, and thus the failure was easily accounted for.

I soon had another suitable case. The patient had been previously hypnotised and cured of spinal neuralgia, insomnia, and dysmenorrhœa. She also suffered from menorrhagia; the periods invariably lasted a week, and were excessive all the time, necessitating complete rest in bed. Suggestions given in the ordinary way having failed to relieve this, I placed the patient in a nursing home, and kept her in the hypnotic trance for three days before and five days during a period. Since then menstruation has been normal in quantity and duration, and, instead of being confined to bed, the patient has been able to bicycle and take other active exercise.

In 1893 I visited Drs. van Eeden and van Renterghem at Amsterdam, and found that in many respects their methods resembled those of Wetterstrand. From 1887 to 1897 they had treated 1577 cases, and of these they have published accounts.

As already stated, the difficulties I encountered in hypnotising and treating certain cases of long-standing nervous disease were the main cause of my going to see the hypnotic work of others. I found, however, that my foreign colleagues experienced difficulties similar to my own; they also had a certain percentage of cases refractory to hypnotic influence, and many in which only slight hypnosis could be induced. I learnt much that was useful, however, not only at the cliniques, but also at the various international congresses of psychology and hypnotism which have been held of late years. Above all, I was taught the value of suggestion in hypnotic conditions so slight as to be hardly distinguishable from the normal, and now invariably make curative suggestions from the very commencement of treatment, instead of waiting, as formerly, until well-marked hypnosis had been induced.

It is impossible to discuss the question of theory in a lecture such as this, but I will briefly summarise the more important points:

1. Hypnotism, as a science, rests on the recognition of the subjective origin of its phenomena.

2. The theories of Charcot and the Salpêtrière school are practically a reproduction of mesmeric error.

3. Liébeault and his followers combated the views of the Salpêtrière school and successfully substituted their own, of which the following are the important points:

(*a*) Hypnosis is a physiological condition which can be induced in the perfectly healthy.

(*b*) In every one there is a tendency to respond to suggestion, but in hypnosis this condition is artificially increased.

(*c*) Suggestion explains everything.

(*d*) Despite the fact that the members of the Nancy school regard the condition as purely physiological, and simply an exaggeration of the

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normal, they consider it, in its profound stages at all events, a form of automatism.

These and other views of the Nancy school have recently been questioned by several observers. As Myers justly pointed out, suggestion is the artifice used by the operator in order to excite the phenomena of hypnosis, but does not create the condition on which they depend. The peculiar state which enables the phenomena to be evoked is the essential thing, not the signal which precedes their appearance.

Within recent times another theory has arisen. This, instead of explaining hypnotism by the arrested action of some of the brain centres which subserve normal life, attempts to do so through the arousing of certain powers over which we normally have little or no control. This theory appears under various names: "double consciousness," "subliminal consciousness," etc., and the principle on which it depends is largely admitted by science. Thus William James, for example, says, "In certain persons, at least, the total possible consciousness may be split into parts which co-exist, but mutually ignore each other."

According to the Nancy school, the deeply hypnotised subject responds automatically to suggestion before his intellectual centres have had time to bring their inhibitory action into play. According to the subliminal consciousness theory, volition and consciousness are unimpaired in hypnosis. The subject has gained an increased power over his own organism, and, through the intelligent action of the secondary self, produces results unattainable by the normal will.

The intelligent action of the second self is illustrated by the execution of certain post-hypnotic suggestions. Thus, when one of my subjects was told during hypnosis that she was to perform some specific act in the waking state, at the expiration of a complicated number of minutes, as, for example, 40,825, she generally carried out the suggestion with absolute accuracy. In this and similar experiments three points are to be noted—(1) the arithmetical problems involved were far beyond the subject's normal powers; (2) except when hypnotised she possessed no special faculty for appreciating time; (3) her waking consciousness retained no recollection of the experimental suggestions or of anything else that had occurred during hypnosis.

The question of secondary or multiplex personalities is a fascinating one, but I cannot now follow it further. It is well to remember, however, that William James, the distinguished psychologist, attaches great value to the manifestations of what he terms the "hidden self," and also asserts that a comparative study of subconscious states is of the utmost importance for the comprehension of our nature.

In justice to Braid it must not be forgotten that there is little difference between his later views and those held by William James, Myers, and others who explain the phenomena of hypnosis by the action of an intelligent secondary consciousness. Braid, unlike Bernheim and other members of the Nancy school, had ceased to regard hypnosis as an artificially induced sleep. He had rejected his earlier views as to monoideism and automatism, and held that volition and consciousness were unimpaired. Although he constantly and intelligently employed suggestion, he did not consider that it explained the ultimate origin of hypnotic phenomena. He also recognised the alternating memories of deep hypnosis, the existence of double consciousness, and the increased moral, intellectual and physical powers of the hypnotic state.

The practical side of the subject is of undoubted value. It is true that the time and difficulty often involved in the induction of the primary hypnosis render hypnotism of little use as an anæsthetic, but these objections only tell to a moderate degree against its employment in medicine. Every one cannot be deeply hypnotised, but fortunately profound states are not necessary for the successful employment of suggestion, and the number of persons insusceptible to some degree of hypnotic influence is extremely small. Wetterstrand, as we have seen, only failed in about 3 per cent.

Amongst other diseases, the following may be cited as those in which hypnotism has given good results:—Hysteria, neurasthenia, dipsomania, morphinomania and other drug habits, obsessions, moral perversity and nervous tricks in children. In this country, at all events, hypnotic treatment is rarely tried until everything else has failed, and thus the disease has become well-established and the patient hopeless. Despite this, hypnotism is frequently successful. The following is a typical case:—Miss X—, *grande hystérie* of the true

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Salpêtrière type. After varied treatment the patient passed four months in the National Hospital, Queen Square, where subcutaneous injections of hyoscine, and large and frequently repeated doses of chloral, bromide, etc., were administered, her condition meanwhile becoming distinctly worse.

On June 18th, 1900, she was discharged from the hospital and came under my care. She was unable to walk; there was a contracture of the muscles of the left leg, constant clonic spasms of the head, trunk, and extremities of the left side, together with frequent and violent generalised convulsive attacks of the true Salpêtrière type. The patient commenced to improve almost immediately under hypnotic treatment, and three weeks later all morbid symptoms had disappeared. Since then she has been actively engaged in work, and her health is excellent. No drugs were given.

The value of hypnotism in dipsomania is especially striking, and some of my earlier cases have now remained twelve years without relapse, this even being true of those who had been confirmed drunkards for years, and who had submitted to long periods of restraint in retreats without benefit.

Further, and the point is an important one, the employment of hypnotism by medical men acquainted with the subject is devoid of danger. Forel asserts that in no single instance has the induction of hypnosis caused mental or physical harm, either in his own practice or in those of Liébeault, Bernheim, Wetterstrand, van Eeden, de Jong, Moll, and other followers of the Nancy school. My own experience is in accordance with this statement, and I have never seen any unpleasant symptom, even of the most trivial character, result from hypnotic treatment.

If any of you would like to see hypnotic work on a large scale, I would recommend a visit to Bernheim's hospital wards or to the cliniques of Wetterstrand and van Renterghem.

If you wish to study hypnotic literature, begin with Moll's 'Hypnotism' and Bernheim's 'Suggestive Therapeutics.' I do not agree with these authors in every detail, but the former gives the best scientific account of the subject, and the latter the best therapeutic one, yet published. These two books by no means exhaust the list. Max Dessoir, in his bibliographies of modern hypnotic literature, refers to some eleven hundred works by about eight hundred authors. There are also various medical journals devoted entirely to hypnotism, the two most important being the 'Revue de l'Hypnotisme' and the 'Zeitschrift für Hypnotismus.' Some day I, too, hope to publish a book on the subject, and have already written various articles dealing more fully with the theoretical points I have referred to. Among these I would cite "On the Evolution of Hypnotic Theory," 'Brain,' part lxxvi, 1896, and "Hypnotic and Post-hypnotic Appreciation of Time; Secondary and Multiplex Personalities," 'Brain,' Summer Number, 1900.

Now that the reality of the facts of hypnotism has been so abundantly proved, and successful simulation rendered impossible by the production of objective phenomena which no one can simulate, further scepticism and indifference seems hardly conceivable. How be indifferent to a science which explains so much of what is mysterious in history, which supplies the psychologist with a genuine experimental method, and furnishes such aid to the study of cerebral physiology? How neglect an art by means of which pain of mind and body can be relieved, and a large number of diseases cured? By means of hypnotism the historian finds the whole subject of magic, oracles, sybils, and the like illuminated with a vivid light.

To the psychologist hypnotism gives a harmless and painless means of intellectual and moral vivisection; while the physiologist, by its aid, can produce anæsthesia and other sensorial conditions, and regulate or modify the secretions, excretions, and other functions of the human body, which the waking will is incapable of influencing.

While those who study and practise hypnotism differ widely in their theoretical explanations of its phenomena, what is common to all, as Professor Beaunis states, is the belief in its future—the profound conviction that this science, so ridiculed, constitutes one of the greatest advances of the human mind and one of its most precious possessions.

An exhibition of appliances for medical instruction will be held from May 31st to June 8th in the Royal Academy, 38, Unter den Linden, Berlin. Dr. Kutner, 13, Elssholz Strasse, is secretary.

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THE TREATMENT OF MALIGNANT STRICTURE OF THE ŒSOPHAGUS.*

By F. J. STEWARD, M.S., F.R.C.S.,

Surgeon in Charge of the Throat Department, Guy's Hospital.

MR. PRESIDENT AND GENTLEMEN,—I propose to deal this evening as far as possible entirely with the treatment of malignant stricture of the œsophagus; I shall have, however, to refer incidentally to some points in connection with the diagnosis and symptoms of this disease. It is convenient, in the first place, to consider the treatment in two stages: first before, and secondly, after signs of involvement of the air-passages have arisen. For, as will be seen later, methods of treatment which may be entirely satisfactory in the first stage, become impossible as soon as a communication between the food and air-passages has formed. Other complications besides involvement of the air-passages, necessarily also modify the treatment, but these complications are so rare that they need not be considered in laying down general lines of treatment for these cases.

With regard to the question of diagnosis, I would lay stress on the importance of arriving at a definite conclusion as to the nature of any case of dysphagia at the earliest possible moment, as there is nothing whatever to be gained by delay, and everything to be lost; for, as I hope to prove to you shortly, the most satisfactory and convenient form of treatment, namely, by tubage, can only be carried out if the diagnosis is arrived at in the early stages of the disease.

In any case of persistent and increasing dysphagia, then, a bougie should be passed in order to ascertain whether stricture of the œsophagus is

* Read before the Watford Medical Society, February 14th, 1902.

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present. At the same time the following three points should be decided, if possible:—(1) the position of the growth; (2) the degree of obstruction; and (3) the extent of the growth. In order to do this, the mode of procedure that I adopt is as follows:—I first take one of the largest sized bougies, the pattern I prefer is the olive headed, which is made of gum elastic with an olive at either end, the olives being numbered and of different sizes. If, as is usually the case, this bougie will not pass, I note the point at which it is arrested, and measure off the distance of this point from the teeth; this, of course, gives the *position of the growth.* I now proceed exactly as in a case of stricture of the urethra, trying smaller and smaller sizes till one passes through. This will denote the size of the stricture, and the *degree of obstruction.*

The third point—the extent of the disease—can be ascertained on withdrawing the bougie that has passed the stricture, for the base of the olive can nearly always be felt to be gripped slightly when it reaches the lower margin of the growth. If the distance of this point from the teeth is then measured off, the difference between this and the first measurement will give the vertical *extent of the disease.*

Occasionally two distinct points of stricture will be encountered at a variable distance from one another. These two points usually denote the upper and lower edges of the growth which tends to ulcerate most markedly in the middle portion of its extent. Rarely, however, there are two distinct growths present.

The question which I expect is now in the minds of most of you is: "What is to be done if no bougie will pass the stricture?" The best plan I think is as follows:—the patient is put to bed, allowed nothing by the mouth but a little water, fed entirely by the rectum, and given a small amount of opium.

The result almost invariably is, that after a few days of this treatment, the spasm and congestion, which have been brought about by the patient's frequent and ineffectual attempts at swallowing, are diminished to such a degree that a bougie can be passed.

If, however, the smallest bougie of the olivary pattern will not pass, a soft silk-web feeding tube of small size can generally be got through, because it is less rigid and the passage is sometimes tortuous.

Finally, there remains a very small percentage of cases in which all these plans fail. These cases can only be treated by gastrostomy.

TREATMENT.—I shall now consider the different methods of treatment separately. These are five in number, namely:

1. By bougies.
2. The short tube.
3. The long tube.
4. Gastrostomy.
5. Excision.

1. *By bougies.* — As long as the patient can swallow solid food in any form, the only treatment necessary is the passage at intervals of the largest sized bougie that will pass without force through the stricture. As a rule, it is wise to pass a bougie twice a week, but in many cases, particularly in the slower forms of growth, the *weekly* passage of a bougie will be quite sufficient.

FIG. 1.—Symonds' short tube.

To give you an instance, I have now a patient attending my out-patients at Guy's, who is æt. 72, and who had had gradually increasing dysphagia for three months when he first came for treatment six weeks ago. It was found then that he had a stricture 13½ inches from the teeth, which admitted a No. 19 bougie. A bougie of that size has been passed every Friday, and he can still swallow finely divided solid food.

As soon, however, as the patient becomes restricted to fluids, tubage in some form should be resorted to, before the rapid contraction which now usually occurs prevents the passage of a tube of suitable size.

2. *The short tube.*—This mode of treatment was perfected some years ago by Mr. Symonds, and the type of tube usually employed is the one suggested by him, and which is usually known as the Symonds' tube. As you see, it is made of gum

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elastic on a silk web. Its upper end, which rests on the upper margin of the growth, is funnel-shaped, the lower end being closed and provided with two large eyes. The tubes are made of different lengths, the length used being dependent upon the vertical extent of the growth. The shorter the tube that will answer the purpose the better, because the liability to become blocked will vary directly with the length of tube. Too short a tube must not be used, however, because the eye would be liable to be blocked by the growth.

The tube is threaded with strong silk, which is brought out of the mouth and fastened round the ear.

The Introducer.—The most convenient form I think is the one I have here, which consists of a whalebone guide, which serves to stiffen the tube and prevents it from coiling up in the œsophagus. As you see, there is also a sheath which fits the funnel at the top of the tube, and thus gives further control.

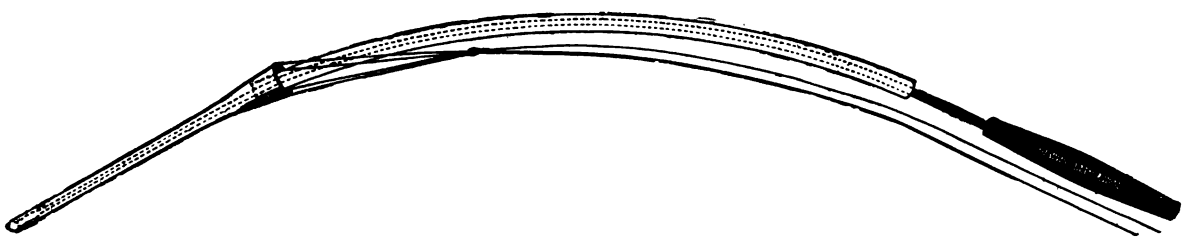

FIG. 2.—Short tube mounted on the introducer.

It is not, however, necessary to use a special introducer at all, as a piece of fairly rigid copper or iron wire will serve the purpose quite well, and, in fact, a copper wire guide was, I believe, used by Mr. Symonds when he first employed these tubes.

The Introduction.—We will now consider the introduction. Having ascertained the distance of the stricture below the teeth, and also the extent of the growth and the size of the lumen, a suitable tube is chosen and threaded with silk.

The silk should now be knotted at the point that will be about an inch inside the teeth, and a piece of fine rubber drainage-tube about two inches long slipped over the silk up to the knot, and the silk again knotted beyond it. This will protect the silk from being frayed out by the teeth or bitten through.

Having now mounted the tube on the introducer it is passed down the œsophagus, the patient sitting with the head well thrown back, and the silk being held taut so as to prevent the tube slipping off the guide. If the tube is not actually felt to enter the growth the knot on the silk will indicate when this point is reached. The tube must now be slowly and steadily passed through the stricture, and on until the funnel is felt to be arrested by the growth. The silk is then released and the introducer withdrawn. The patient should now drink a glass of milk in order to make sure that the tube is clear, and that no piece of growth or food has been picked up by the eye.

The effect of the tube is not only to allow the free passage of fluid nourishment, but also in most cases to bring about a certain amount of dilatation of the stricture. If therefore a No. 8 or 10 tube has been introduced in the first instance, this should be removed at the end of a week and a larger size introduced, until, if possible, a No. 14 can be passed. It is not necessary, as a rule, to pass a larger size than this.

The length of time that a tube can be left in position varies in different cases. Provided, however, that it does not become blocked, that it does not cause pain, and that evidence of involvement of the air-passages does not arise, the tube can be left in indefinitely. As a rule, however, it will require changing about once a month, although occasionally no change is needed for two months, or even more. Should it become blocked at any time the tube must, of course, be removed, cleaned, and reintroduced, the silk being carefully examined before doing so.

If no perforation of the air-passages occurs the tubes may be continued for the rest of life, the patient being free from pain and discomfort, and able to take plenty of nourishment during the whole time.

The change produced by the introduction of

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a short tube is often very striking, the patient usually gaining weight and strength, and often being able to keep about and attend to his business till very near the end of his days. If, also, as is often the case, the tube is introduced after swallowing has become almost an impossibility, the delight of the patient at being once more able to drink freely is a sight well worth seeing.

The following case will further illustrate many of the above points. A man æt. 64 was admitted under my care into Guy's Hospital on June 4th, 1900. He had suffered from increasing difficulty in swallowing for six months, had been unable to swallow solids for the last two months, and on admission could only swallow liquids with great difficulty and very slowly. He was emaciated, cold, and extremely feeble, and, in fact, was practically dying of starvation. I found a stricture twelve inches below the teeth, but the smallest bougie would not pass; I therefore fed him by the rectum, kept him warm in bed, and gave him a little opium for four days. At the end of that time I passed a No. 9 bougie fairly easily. A No. 8 short tube was then introduced without difficulty. He took plenty of nourishment, and rapidly improved. The tube was changed on June 16th and a No. 10 introduced, and again on June 24th and a No. 12 put in. He then left the hospital with the tube in position. In the next few weeks he gained 10 lbs. in weight, and was able to return to his work. The tubes were changed occasionally, and continued perfectly satisfactory until his death, which occurred suddenly from hæmorrhage, no doubt due to perforation of the aorta, in November, or five months after tubage was commenced.

Other cases might be cited, but I think this one serves sufficiently to illustrate the main points. One very exceptional case I might mention, however. It was a man who worked at Woolwich Arsenal, and who attended Mr. Symonds' Throat Out-patients for over twelve months, wearing a tube and doing his work the whole time.

I must refer now to the *accidents* that may occur in connection with these tubes. The first is blockage with food, that I have already mentioned. In the next place, the silk may break. This must be a very rare accident, because silk is very strong and durable, and although I have now seen a good many cases treated I have not yet seen such an accident happen. It has, however, I know, happened to others. In itself the breaking of the silk does not matter much, but usually the end gets washed into the tube and blocks it.

This accident may be dealt with in two ways. In the first place, attempts may be made with a piece of hooked wire, or even with forceps, to catch the loop and withdraw the tube—this has been done successfully. Or the tube can be pushed through the stricture into the stomach with a bougie. No harm results, the tube being either retained in the stomach or partially dissolved and eventually passed. It is not possible to treat every case in this manner, for in some the tube causes irritation, pain, or coughing, and cannot be borne, and in others the involvement of the air-passages in the growth makes it necessary to resort to other modes of treatment.

The cases in which the tubes cannot be borne are those in which the growth is situated very high up or very low down. If the growth is situated at the level of the cricoid the hard tube causes ulceration, pain, and coughing as a rule. If it is at the cardiac end of the stomach the canal is frequently tortuous, so that no tube can be introduced; in a few of these cases, however, a short tube can be used. It is therefore well to make a trial of this method in the first instance.

3. *The Long Tube.*—The next method of treatment is that by the long, soft tube. This is suitable in two classes of cases which cannot be treated by the short tube. The first class consists of cases in which the short tube causes pain, or coughing, from irritation of the larynx; and the second class, of cases in which extension of the disease has led to a communication being formed with some portion of the air-passages, either trachea, bronchus, or lung. Here the short tube can be no longer used, because it is necessary to prevent food reaching the air-passages. The tube consists of pure rubber; in fact, a piece of ordinary drainage-tube closed at the lower end and containing a large eye in the side. This can be either introduced on the whalebone guide or upon a guide of wire. The largest size that will pass should be used, because the pressure of the growth flattens the tube, and so tends to lessen the lumen. It is necessary to use a pliable, thin-walled tube for two reasons. The first is to prevent irritation, the second to ensure the widest possible bore for feeding purposes. The tubes I have here have been made for me by

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Messrs. Meyer and Meltzer; they serve their purpose extremely well, but only differ from the form of the tube used by Mr. Symonds as regards the lower end, which is sealed instead of being simply sewn up. The tubes are perfectly comfortable, cause no irritation, and prevent food passing into the air-passages. They have, however, two disadvantages, in that the saliva cannot pass down, so has to be expectorated, and that the growth tends, as I said before, to compress the tube, and so obstruct it.

The following case serves further to illustrate this plan of treatment:—Mrs. V—, æt. 33, I first saw in consultation with Dr. Snell, of Wandsworth, at the end of August, 1901. She had dysphagia, which had been gradually increasing for fourteen months, finally becoming absolute a few days before I saw her. During the last week a large brawny swelling had appeared in the neck, and the temperature had been raised. She was extremely ill, the swelling, which fluctuated below, reaching from the level of the thyroid cartilage to the sternum. I made a small incision into the fluctuating part, and let out a quantity of foul gas and a little pus. After this she improved for a time and could swallow liquids, but ten days later the dysphagia again became absolute, and all food passed through the wound in the neck. She was therefore admitted into Guy's Hospital under my care on September 6th. Under an anæsthetic I succeeded, after considerable difficulty, in passing a soft tube through the stricture, which was at the level of the cricoid; I also opened up the cavity in the neck, which was not draining satisfactorily. She was then given plenty of fluid nourishment, and rapidly improved, leaving the hospital with the tube in position on September 28th.

She was later admitted into Mr. Lucas's ward with difficulty in breathing, for which tracheotomy had to be performed. She was, however, fed by means of a long tube until her death, which took place on November 30th, practically three months after tubage was commenced.

4. *Gastrostomy.*—I will now consider the question of gastrostomy. I should only perform this operation where tubage in any form is impossible, because the operation is a very fatal one in these starved patients, and, moreover, the condition of the patient is certainly not as satisfactory or as comfortable as it is when a feeding tube can be used. To my mind there is no comparison between the two conditions, in the one the patient runs no risk from operation, can drink freely, can swallow his saliva, can get about, and often follow his employment till almost the end of life; whereas if a gastrostomy is performed, there is not only the grave risk of the operation, but the discomfort of a fistula, which almost always leaks to some extent, the mental distress caused by an unnatural mode of feeding, and the misery of being unable to swallow the saliva. Nevertheless, it is sometimes the only plan possible, and should then certainly be resorted to if the patient is not too far gone. Of the many methods that have been devised I much prefer Franck's, because it is simple, easy, and rapid of performance, provides an efficient valve, and enables feeding to be commenced at once. The following case will serve to illustrate further some of these points:

William W—, æt. 53, was admitted into Guy's Hospital on August 22nd, 1901. He had suffered from dysphagia for four months, and a month before admission had been seen by Mr. Symonds, who found a growth at the cricoid level, and introduced a long soft tube. A few days before admission the tube had become blocked, and was removed by the patient.

On admission, he had complete dysphagia, and in spite of repeated attempts, I failed to pass any kind of bougie or feeding tube. It was therefore decided to perform gastrostomy. This I did on August 23rd by Franck's method, the operation taking about fifteen minutes. Feeding was commenced three hours after operation, the patient rallying well and rapidly improving. He left the hospital on September 15th. At this time the valve acted well, no leakage occurring, except immediately after the introduction of food, when a small amount came away. He was, however, unable to swallow any saliva, and, moreover, had a troublesome cough, and was consequently expectorating constantly.

5. *Excision of the Growth.*—Finally, I must mention the question of excision. The possibility of this must be very rare, and I only know, in fact, of one successful case. This was performed by Czerny in 1877, and the case is so interesting that I will describe it.

The patient was a woman æt. 51. Czerny made

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an incision from the level of the hyoid bone down to the sternum along the anterior border of the left sterno-mastoid. The omo-hyoid muscle was divided, the thyroid body pushed inwards, and the œsophageal tumour, which could then be reached, was carefully dissected out, 6 cm. of the gullet being removed, and the lower cut end attached to the skin. All went well, and the woman soon learned to introduce a catheter through the fistula and to feed herself in this manner, and five months after operation she was in perfect health, without a trace of recurrence.

The Treatment of Erysipelas in the "Red Room."—Herman Krukenberg reports the treatment of eighteen cases of erysipelas by means of red light, in a room especially provided for this purpose. The results obtained were very satisfactory in that the duration of the disease was greatly shortened, in thirteen being less than two days; furthermore, systematic symptoms, such as fever and headache, were rarely observed. No general or local medication was given, except in three cases, in which alcohol was locally applied. In those cases in which the local lesions were sharply defined at the beginning of the treatment such a definition began to vanish after a few hours in the "red room," and the swelling markedly decreased. Four of the cases relapsed after removal from the red light, but were speedily cured by a continuance of the treatment. The author does not think that red light in itself possesses any curative influences over erysipelas, but that the benefit derived therefrom is due to the exclusion of the chemical rays of the sunlight. The streptococcus of erysipelas does not appear to be materially injured by daylight, and the explanation of the benefit derived from red light is probably to be sought in the fact that the chemical rays irritate the skin, and make it particularly susceptible to the action of the streptococcus. It is possible that the beneficial results obtained in the local treatment of erysipelas with ichthyol, iodine, and various plasters, is principally due to the exclusion of the light thereby. This is shown by the speedy recovery of a case of facial erysipelas, which was treated in a dark room, in which, when illumination was necessary, a red light was used.—*Medical Record*, April 26th, 1902.

THE DETECTION OF PHTHISIS IN ITS EARLIEST STAGE.

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GENTLEMEN,—The diagnosis of phthisis is never difficult when the disease is well advanced, by which I mean that the lung is more or less solid, that the tubercular deposit is softening and in process of cavity formation. Under such circumstances the dulness on percussion, the bronchial breathing, the prolonged expiration, together with the presence of added sounds, form a complex of physical signs which not even a tyro could overlook. It is rather the early diagnosis of the disease to which I would this afternoon draw your attention; that stage, which it is of the most vital importance, should be recognised without fail, as it is during this stage alone that treatment is really of any avail, so far, at all events, as relates to entire recovery from the disease.

What, then, are the very earliest *signs* of phthisical conditions of the lung apex? On inspection it will be probably found that the affected side moves somewhat less freely than its fellow, it lags behind as it were, its movements being limited as regards its excursion both upwards and outwards. On palpation you will find that the lack of movement already detected by the eye is appreciated and confirmed by the hand.

On percussion a high-pitched note will be obtained over the clavicle on the affected side, as compared, of course, with the sound lung, and if both lungs are involved, there is, nevertheless, certain to be an appreciable difference between the response to percussion on the two sides. The percussion referred to is, of course, that of the clavicle, and only over its inner half. It may seem superfluous to say this, but experience has taught me that this is not the case, as I have over and over again known the manipulation to be carried out over the outer half of the clavicle under which no lung lies.

The auscultatory signs at this stage of the disease are often very slight. There may be nothing more than harshness of inspiration as compared with the other side, and some undue lengthening of the expiratory murmur. Again, at the extreme end of

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inspiration, a faint unmusical sound, a crackle, is often present. This is a most important sign. This crackle will often become multiple after the patient has coughed and when he takes a deep breath, and no opinion should be given in a doubtful case unless this effect of coughing has been sought for.

Another most important evidence of early phthisis is an interrupted, broken inspiratory murmur. To this most characteristic sign the name of "cog-wheel" breathing has been appropriately given. It is impossible to over-estimate the value of this sign of early consolidation, and due weight should always be given to its presence, even in the entire absence of other abnormal physical signs. The interruptions may be three or four in number, or they may be one only. So far as I know a single interruption of the inspiratory sound has the same significance as several breaks in the same.

I must now mention a most important point in the diagnosis of early phthisis. The examination of the chest must be by no means limited to the apex of the lung in front; it is most essential that the suprascapular region be subjected to a close and careful scrutiny. Very often, indeed, physical examination will reveal evidence of early phthisis in this situation when nothing abnormal can be found in the clavicular region. The moral is, of course, that the suprascapular region should be most thoroughly and carefully examined in every case, and an opinion withheld till this has been done.

I need say but little as to the physical evidence of lung consolidation, which depends upon the increased density of the affected lung. Naturally, as sounds are conducted better the more solid the conducting substance, there must be increase of vocal resonance and of vocal fremitus. Further, in early phthisis it is quite usual to hear the heart sounds unduly loudly over the affected area. This should be recognised as a sign of some importance when it is well marked.

So much, then, for the ordinary signs of early phthisis. I need scarcely say that in every case it is not safe to judge by signs alone, as I shall have occasion to further consider presently, and it should be the invariable rule that the history of the illness and the family history of the patient be most fully considered, together with the symptoms complained of. In this way only will it be possible to avoid the danger of making very serious mistakes.

A few weeks ago a patient æt. 22 presented himself in this room complaining of severe cough, with some expectoration, and of febrile symptoms. He stated that he had only been ill about a fortnight, and had been in his usual health until that time. On examination there was evidence of catarrh of the bronchial tubes, those of large and middle size being more or less involved, and there was also catarrh of the larynx and pharynx. Careful percussion proved the left infra-clavicular region to be somewhat higher pitched on percussion than the right.

A day or two ago a child æt. 2½ was brought to this room with the history of its being well until ten days ago; indeed, she was at school till the commencement of the illness.

At the time mentioned she first complained of cough, and was hot and feverish, but did not take to bed. The previous health was stated to have been always good. On examination marked dulness, bronchial breathing, and abundant expectoration were found beneath the left clavicle and for about as far down as the left nipple.

The subsequent history of these two cases was full of interest.

The young man suffering from catarrh came again a fortnight later. The catarrh had practically vanished from the bronchial tubes except at one spot—that, namely, where high-pitched percussion resonance was detected. Here the high-pitched note still persisted, as did also the signs of catarrh. A week later the condition was just the same. It now transpired that there was a family history of tuberculosis, and also that the patient had suffered from a slight cough for six months before the present illness.

When the child was brought again to hospital about four days later a great change was noticeable as regards the physical signs present over the left upper lobe. The dulness was still marked, but the bronchial breathing had quite vanished. Instead of abundant crepitation, which had accompanied both expiration and inspiration, only a few crackles at the end of the latter were audible. The temperature was only 99·6 at the first visit, and at the second was normal.

The diagnosis in the child, of course, was either

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that of phthisis or of an acute upper lobe pneumonia. The history pointed strongly in favour of the latter malady, and the subsequent course of the illness proved that this was the disease actually present.

But in a case such as this the physical signs are perfectly consistent with the possibility of either diagnosis being correct; from them *alone* it would be absolutely impossible to say which of the two maladies was actually before one's eyes. It may be urged that the case was extremely unlikely to be pneumonia, on account of the severity of the symptoms when that disease attacks the upper lobe. I know well that this is the popular view as to such pneumonias, but I must express my entire dissent as to its accuracy. In my experience upper lobe pneumonias are neither more prolonged or severe in their cause, nor are they more liable to be attended with delirium than where the condition affects any other portion of the lung substance. It is, of course, unusual for the symptoms of pneumonia to be so mild as they were in this case, but though unusual, there is no doubt that such cases do occur from time to time, just as ambulatory typhoid is not unknown.

We may therefore say that this child was in reality suffering from acute pneumonia, the prognosis of which is altogether favourable.

To turn now to the other patient, the young man. In this case the temptation to regard the malady as bronchial catarrh was undoubtedly very strong, and had it not been for a generally delicate aspect and slight build one might very well have dismissed the case as such. But as I have said, careful percussion detected a high-pitched note over the left upper lobe in front, and this was a physical sign which could not be hastily overlooked.

The caution exercised was soon justified, for when the bronchial catarrh had cleared up then it was that the physical signs of early phthisis were unequivocally evident.

We see from both these cases the absolute necessity of having a full and clear history of the illness and also of the family history; they also warn us strongly against undue haste in forming an opinion, and they show us in the most striking manner how very unreliable physical signs by themselves alone may be.

But phthisis does not always commence at the apex, and the onset of the malady may be concealed, as it were, under the guise of pleurisy with effusion. The following case illustrates in a striking manner the truth of what I have just stated:

A boy æt. 16 came to me in the out-patient room stating that until just a week before attending at the hospital he had been in his usual excellent health. There had been no antecedent illness, and the family history was entirely free from a tubercular taint.

At the time mentioned this boy underwent a severe wetting, and shortly afterwards he felt acute pain in the right chest, worse on drawing a deep breath. He had also slight cough, accompanied with expectoration and some shortness of breath. In a day or two the cough became only trifling, but the shortness of breath continued. It was on account of the persistence of this symptom that he sought advice.

On examining him I found a considerable pleural effusion on the right side, and in the lateral region on that side there was marked friction.

He came into hospital, and his temperature was found to be 102°.

For a week or ten days the temperature remained febrile, but the condition was as on admission. There was some night sweating. About a fortnight after admission examination of the right infraclavicular region yielded signs which were certainly suspicious; there was distinct loss of resonance on percussion, and the inspiration was harsh and occasionally of the "cog-wheel" character already referred to. Expiration was distinctly prolonged, and after coughing crackling could be detected at the end of inspiration.

Nevertheless, it was perfectly obvious that the pleural effusion was undergoing absorption, as all the physical signs of its presence were becoming less marked.

Three weeks later the effusion at the right base had practically cleared up, but the physical signs of consolidation at the right apex were now absolutely unmistakable. There was marked dulness, the inspiration was very decidedly of the "cog-wheel" type, the expiration was prolonged and the crepitation was abundant, being also greatly increased after cough. His temperature was now normal. This patient, who entered the hospital with pleural effusion, the result of exposure, left it

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with no pleurisy, but with marked phthisis on the same side as the effusion.

This case is one of great importance, showing as it does the insidious onset of pulmonary tuberculosis under the guise of pleurisy with effusion. And the absolute necessity of careful and repeated examination of the apices is never more urgent than when pleural effusion supervenes, apart from a definite injury and in a person presumably healthy.

I have seen many of these cases, and in by far the greater number the effusion has been the herald of phthisis, although the physical signs have seldom come out with the rapidity which was so marked in this case.

There is one caution that it is necessary to be aware of. It is not safe to deduce any inference from the presence of slightly altered percussion note and of bronchial breathing, or prolonged expiration at the apex of a lung when considerable pleural effusion is at the same time pressing on the base of the organ. The pressure of the fluid tends to alter the note detected at the apex, and also, to a certain extent, modifies the natural vesicular murmur in the same locality. But when crepitation is present it undergoes no particular change as the result of pressure on the base of the affected lung.

It must not be forgotten that in exceptional cases phthisis may commence at the apex of the lower lobe of the lung. Not long ago a young man came to me in this room complaining of symptoms pointing to phthisis as their cause, but in whom no physical signs of the disease could be discovered at either apex. On examination of the lower lobe, however, it was found that the upper part of that region on the right side was solid from tuberculous deposit. Of course, in a case such as this it may not be possible at first to exclude a resolving pneumonia; in the instance referred to it was, however, possible to do this from the history of the case.

It is certainly far from common to meet with cases in which phthisis commences elsewhere than at the apex of the lung. Still such do occur from time to time, and it is often of much importance to be aware of this.

The diagnosis of "fibroid" phthisis, that form of the malady in which the lung becomes infiltrated with new-formed connective tissue, is not always easy. In some cases it is particularly difficult to differentiate between this variety of phthisis and malignant disease affecting the lung and pleura.

I recall a case in which the physical signs were consistent with the presence of either of these maladies, but in which the fact of the occurrence of aortic regurgitation rendered the diagnosis of phthisis to be probably correct.

For it must not be forgotten that it is in this form of phthisis that valve murmurs are especially common. In most cases of phthisis the heart is entirely free from any evidence of valve disease, but "fibroid" phthisis differs greatly from the more usual varieties of the affection in this respect.

In the case I refer to, however, there was a most distinct aortic regurgitant murmur, and the other features of the case rendered it probable that cirrhosis of the lung was the disease actually present. A needle introduced into the pleural cavity revealed the presence of blood-stained fluid. This, of course, pointed strongly to the malignant nature of the pulmonary affection. But it must not be forgotten that, although the physical signs pointed to the presence of a certain quantity of fluid, yet they were quite consistent with marked thickening of the pleura and adhesion of the same to the pulmonary substance. This being so, it is obvious that the blood might come from the pulmonary and pleural tissue.

The post-mortem examination proved the case to be one of malignant disease of the lung, together with atheroma of the aorta and incompetence of the aortic valves.

This case is a most interesting and important one, as illustrating the close resemblance there may be between the physical signs of chronic phthisis and those of cancer of the lung.

And this similarity is not altogether limited to cases of *chronic* phthisis. In the more acute tubercular forms the difficulty may also arise, as in the following instance:—A middle-aged man complains of persistent weakness and loss of flesh, but of no other symptoms. Cough is so slight as to be scarcely noticeable. On examination evidence of pleural effusion is found at one or other base, perhaps with some displacement of the heart. Both apices are entirely free from disease.

In a case such as this the first thought is, of course, that the malady is tubercular phthisis commencing as tubercular pleurisy. But there

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has been no cough, no shortness of breath worthy of notice, and, although the symptoms have persisted many months, yet both apices are intact. Now, if on the introduction of the needle of the exploring syringe blood-stained fluid is found, the evidence of malignant disease becomes exceedingly strong.

But even if malignant disease is present it does not follow that fluid of any kind will be withdrawn, then all that can be done is to watch and wait, giving a most guarded prognosis.

In concluding these remarks on the diagnosis of phthisis there is one very important matter to which I must draw attention.

It is not sufficiently recognised that in some cases phthisis heals spontaneously, and this even in subjects who are exposed to the most unfavourable influences. Those who have had the inestimable privilege of observing many post-mortem examinations cannot fail to have noticed the comparative frequency with which scars and puckering of the pulmonary apex, together with thickening and adhesion of the overlying pleura, are met with. If the apex of the affected lung be cut across the edge of the knife will probably grate against calcified nodules, and it will be found that these masses are embedded in the centre of a thickened airless patch of scar tissue, which is surrounded with emphysematous lung.

Now these nodules and these cirrhosed patches are nothing more nor less than the relics of a long past phthisis, a phthisis that has healed spontaneously.

We must not claim the result as a theurapeutic triumph, as, unfortunately, experience teaches us that the disease has amended under the most insanitary and squalid surroundings.

I do not at the present moment, however, wish to dwell on this aspect of the case, but desire to point out that such lesions as these do give physical signs. They cause high-pitched note, harsh or obscure breath-sounds, with prolonged expiration, and, not seldom, crumpling or creaking sounds which may be mistaken for crepitation. Hence the incautious and rash observer may draw wholly unwarrantable inferences as to the meaning and nature of these physical signs.

How are mistakes to be avoided? Very simply. By never under any circumstances making a diagnosis solely from physical signs, but always by obtaining every possible information concerning the history, previous history, and family history of the patient. This is a truism, but experience, I regret to say, has taught me that it is one that needs to be constantly repeated.

DEBATE

ON

CANCER: ITS ORIGIN, NATURE, AND GENERAL PRINCIPLES OF TREATMENT.

Held under the auspices of the Chelsea Clinical Society, at the Jenner Institute of Preventive Medicine, March 11th, 18th, and 25th, 1902.

Dr. CHARLES MORRIS, C.V.O., President, in the Chair.

(*Continued from page* 59.)

DR. BEATSON.—Mr. President, ladies, and gentlemen, my first duty is to thank your Council and the members of this Society for what I regard as a great honour in being asked to be present and to take part in the debate. When I received the invitation, the interpretation I put upon it was not that anything I had contributed was of special value, but that you simply recognised that in me you had an interested worker in connection with a subject of the very greatest importance and the very greatest interest. I accepted the invitation because I felt I would like to hear what others had to say, and I have not heen disappointed in the opening address by Dr. Galloway. The facts he has brought forward are all of them of the greatest importance, as bearing upon the subject. Again, I felt that a Society such as this, in having a debate of this character, did a good thing. In a problem of this kind, presenting such great difficulties, it is only by a united effort that we can in any way solve the problem.

The first thing, in carrying on a discussion of this kind, is for us to clearly understand what it is that we are talking about. In the present day it is a proper thing to know that we are all worshipping in the same tabernacle, and that we have not each got a little kirk of our own. With regard to the terms cancer and carcinoma, in speaking of them I refer to a morbid process which begins in a situation where epithelium is present in the body, and results in a structure made up of epithelioid cells. The characteristic of that structure is that the cells continue to multiply and increase, that they invade the surrounding tissue, that they travel, as Dr. Galloway has told us, along the lymphatic vessels, and pass to the lymphatic nodes, and in a large

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number of cases that cells hive off from the primary growth and form secondary deposits in distant organs or parts of the body. That is the meaning which I attach to the terms cancer and carcinoma. At the same time, in a discussion such as this I think it is of great importance that, although speaking of one particular process, we do not lose sight of the whole subject of tumour formation. If we have a theory which explains only one form of tumour, it is, in my opinion, defective and unsatisfactory. In connection with that point there are one or two statements which I think may be introduced here. First of all we have to remember that there is undoubtedly a connecting link between malignant and benign growths. This arises from the fact that both are composed of tissues of the body, and that these tissues have all had a common parentage; they have all sprung from *two epithelial cells*, for the male and female cells by which this body of ours is created are, to all intents and purposes, epithelial cells. As a consequence of that, no matter what the ultimate history of a cell may be, it has, at one time in its life-history, had an epithelial form. In the next place differentiation does occur, so that we have the character of the cell altered and its epithelial form lost. This differentiation varies in amount. In some cases it is only very slight, purely functional, while in other cases it is very marked, and it results in the formation of tissues of a higher type. These specially differentiated tissues, as long as they retain their specialised form, breed true, that is to say, epithelial cell produces epithelial cell, and connective-tissue cell produces connective-tissue cell. Further, another important point is that differentiation lessens the proliferating power of cells. A daily proof of this is seen in connection with the amount of regenerating power shown by different tissues in the cases of wounds and injuries. While proliferation takes place actively and freely in the lower tissues, like epithelium and connective tissue, those of the higher type, such as muscle, are scarcely reproduced. Differentiation, therefore, lessens the proliferating power of cells. Again, certain facts seem to me to show that this differentiation only masks that epithelial type to which I have referred, and, what is very important, that the less highly specialised cells may lose that specialisation and revert to their primitive epithelial form. In no other way can we explain a fact which is familiar to all surgeons of experience, viz. that we have benign tumours suddenly becoming malignant. If, then, a benign tumour may become malignant, if it has, in other words, the power of reverting to a lower type, we must not deny to the tissue it represents the same power.

The conclusions I would draw from these facts are these: (1) That, if the influence or influences—because there may be more than one—that cause tumour growth come into play, the effects will vary with the tissue involved; (2) if it is a highly specialised tissue at the time it is attacked, then we will have a limited growth, in other words, a benign tumour. If it is a very little differentiated tissue, as, for instance, epithelium or connective tissue, we have a great proliferation, an indefinite growth, in other words, a malignant tumour; (3) that malignancy or simplicity of tumours depends on the proliferating power of the cells attacked by the exciting cause of tumour growth, and that this proliferating power is to be measured by the amount of specialisation to which the cells attacked have attained, or that they possess at that particular time, for they may have lost some of it and come down lower in the scale; (4) that the mere division into malignant and simple growths is short-sighted, and, as Virchow, I think, long ago pointed out, merely to take that as a basis of classification would be as unscientific as in botany to divide plants into poisonous and non-poisonous, edible and non-edible.

We have, therefore, always present in the body one very important factor of tumour formation, namely, a proliferating power in its cells. It is also clear that this power varies with the natural differentiation of the tissues, and that this differentiation may be lost in amount, so that a tissue may be brought to a lower standard and thus acquire increased proliferating power. With this one factor in tumour growth present in the body a very important element in that process exists, and the only other factor required to explain tumour growth is the *necessary stimulus*. Of this I shall speak later.

I should like at this point briefly to draw attention to the similarity which exists between cancerous and early embryonic tissue. As I have said, the body springs from two epithelial cells. For a time those tissues which are formed from them are of the same structure as cancerous

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growth, and it is only in connection with such embryonic tissue and its power of development that we have anything approaching the proliferating power of malignant tumours. Again, I should like to note, also in connection with embryonic development, the relation of malformations to neoplasms. If we are right in the theory that we hold about uniovular twins, it is peculiarly a pathological process, as are also joined twins, instances like those which have been of such interest to our Gallic neighbours and some members of the profession over there, the Siamese twins as they are called. Then, again, we have children with extra limbs, or with simply incomplete bodies attached to them. The two-headed nightingale of my younger days is an instance of that. So, also, we have children with sacral tumours and teratoma, and, lastly, we have children with dermoids. We have thus, in connection with what is practically embryonic life, a complete series of gradations bearing on the question of tumour formation.

Now you will expect me to give you, in speaking of such a subject as the nature of cancer, my views as to whether it is a local disease or whether it is a local expression of a general disease. I am not going into that question. All I will say is that I have made up my mind that it is purely local in its initial stage. It commences in alterations in a cell or cells of the parts affected. What exactly that alteration is I shall afterwards have a word to say upon.

We next come to what really is the crucial point in connection with this tumour question, a consideration of the factor or factors which start the local cell proliferation, in other words, the causes of tumour formation. The natural division of such a subject is, causes from without and causes from within. Of those from without, the parasitic theory of which Dr. Galloway has spoken to-night, and given us very useful illustrations, is the only one which requires notice, and I shall only briefly allude to it. As regards its nature, the general feeling is that while bacteria may be the cause, they are not so likely to be so as those protozoa or yeast fungi which Dr. Galloway referred to. This view has been chiefly supported, I think, by the discovery of those so-called cancer bodies of which we have just had such excellent illustrations projected on to the screen. Now, it would be impossible, in a discussion such as this, to take up all the points connected with these bodies, and I cannot go into the explanation of their meaning, but I am of Dr. Galloway's opinion that they are simply cell degenerations. The chief points that weigh with me against the parasitic theory of malignant growths are as follows:—First of all there is that one fatal objection, the question of congenital tumours. Secondly, there is what we observe in secondary growths. In the secondary growths, if they are due to a parasite, the parasite should travel there too. That is not the case, as has been demonstrated. Thirdly, there is the absence of toxins in the body, and that to me is a very important point. Any one who has had experience of cancers, and especially of the early stages of them, is satisfied that the system generally is not poisoned by anything of the nature of a toxin, such as one would expect to result from a parasitic growth. Therefore, I am quite strongly of the opinion that in that view we have not got the solution of the exciting cause of tumour formation.

I have a strong belief in an intrinsic cause which may explain the commencement of local cell proliferation, and I may say to you at once that it is based upon the results which have been obtained in oöphorectomy for mammary cancer. That procedure has been discussed from a therapeutic point of view, that is to say, the question of its value as a remedial agent. That, no doubt, is a very important point. To me it is not so much so as the explanation of the fact that under that procedure masses of cancerous tissue have disappeared. Now I make that statement not simply upon my own work. Others have tried it, and have obtained the same results as I have. Therefore it is a clinical observation which cannot be altogether ignored, and I believe it is of very great importance in connection with the subject of tumour formation. Various explanations have been put forward of the fact that if you perform oöphorectomy in a case of mammary cancer the cancerous tissue does disappear. One reason that has been given is that it is owing to a diminution of the vascularity of the part. Inasmuch as the ovary influences the part where the mamma lies, its removal lessens its vascularity, and accordingly the tumour disappears. Another view is that the oöphorectomy furnishes an increased power of resistance to the other body cells. And, lastly,

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some have asserted that it is the mere laparotomy itself which brings about the change. Well, gentlemen, I believe these explanations are quite inadequate. I am satisfied that we require to find something of a factor much more potent, and one which will explain it much more satisfactorily.

In my original paper I suggested that the explanation was to be found in facts connected with the testes and the ovaries, and I put forward two channels by which the ovaries might be the exciting cause of mammary carcinoma. First of all, they might be so through the agency of migrated ovarian cells, or, secondly, by an altered secretion. Now a word as to the migrated ovarian cells. When we think that there is possibly a daily discharge of ova, only a few of which enter the Fallopian tubes, the rest being probably lost in the peritoneal cavity, the possibility of this migration of cells is not a very far-drawn fact. Then with regard to the occurrence of these cancer bodies which have been illustrated to us to-night, they raised the question in my mind, Are they germinal cells? Because we must remember—and it is a very interesting fact—that one of the first animal cells to be discovered under the microscope were the spermatozoa, and for a matter of 200 years they were thought to be parasitic animalculæ, in the same way as these cancer bodies are considered by many pathologists to be of a similar nature. It was only well on into the last century, I believe, that the real nature of spermatozoa, as seen under the microscope, were demonstrated by Kolliker. With the view of testing this question it was approached from two points: first of all by microscopical examination. Films were made, and in all these cases careful observations were made as to whether anything in the nature of extruded polar bodies could be detected. In one or two cases structures that stained green were noted, apparently leaving the cells, but they might have been, and probably were leucocytes. So, as regards the microscope, nothing confirmatory could be obtained. Next a series of experiments were made consisting of the inoculations of tissues, ovarian and testicular, into rabbits. Six experiments were done, the results of which I published in the 'British Medical Journal.' In connection with those nothing confirmatory was found. I felt then that I had no proof in support of my theory of migrated ovarian cells, and that it was one that I must at present abandon. Then came the question of altered secretion. With the view of testing that, the ovaries I had removed in oöphorectomy for cancer of the mamma were chemically examined, and no confirmatory evidence was found there. Consequently, with regard to the two original views which I put forward, I was bound to say I was wrong, or, at all events, that I had no confirmatory evidence. Was any other explanation available? It occurred to me that there was, and that it lies in what happens in connection with the function of the testes and ovaries. Briefly, both these organs have to prepare cells that will perpetuate the species. The testes do this by forming spermatozoa out of epithelial cells by a process of cell division, and the ovaries by carrying out those maturation phenomena which fit certain of their epithelial cells for fecundation. Both processes exhibit great cellular activity. By what agent are these changes accomplished? What is the stimulus that sets up the cellular division and in the case of the testes produces cellular activities which lead to extrusion of the spermatozoa? What is it that excites those polar bodies and the preparation for fecundation? It must be a *secretion* of some sort, and that being so, where is it elaborated? Is it elaborated by the testes and ovaries themselves? Or is it elaborated by all the tissues of the body under the stimulus of these organs in accordance with the old Hippocratic idea? Are the testes and ovaries merely the instruments for carrying out the work done by the rest of the body? In other words, is the fructifying power which gives the spermatozoa or the female ova their procreative power a product of the whole body or merely a local manufacture? I am inclined to the view that it is furnished by the whole of the body, and such an explanation is not altogether unsupported by facts. Let us take what has been observed in connection with the thyroid gland, one of the so-called ductless glands. It was for a long time considered that it did nothing in the way of secretion or excretion; but we know now, by clinical observation and by other experiments, that the thyroid has a very important duty to perform, and that its function is connected not with elaborating any special secretion itself, but by dealing with the secretion which is poured out by the whole of the tissues. So much is this the case that if atrophy of that

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gland takes place we have a general affection of the tissues, as in myxœdema, while if the gland is completely extirpated we have effects disastrous to the whole fabric of the body. The subtle influences that the testes and ovaries exert on the general nutrition of the body, and the profound impression made upon it by their removal, are well known, and proof of it is seen in eunuchs, and also after oöphorectomy for many ovarian diseases. What, then, is the actual relationship between the rest of the body and testes and ovaries, supposing these conditions to be such as I suggest? When these organs dwindle with age, or become functionless, what happens to this body secretion? Does it diminish also under the lessening influence of these organs, and does it cease entirely with their complete atrophy? Or does this body secretion continue after they are functionless, or are rendered functionless by disease, and is again taken up by the tissues themselves? Lastly, when do these organs lose entirely their influence over the functional activity of the body? You can understand it is impossible to answer these questions in our present state of knowledge, and consequently reliable conclusions are difficult. Facts, however, seem to me to point to the tissues ceasing to give up this fluid on the removal of testes and ovaries.

We can now judge, I think, of the bearing these views have on the causation of the cancerous process. I need hardly point out that the existence of such a stimulating or fructifying fluid in the body tissues furnishes an explanation of many observed effects, and opens up also other possibilities. Of the greatest importance is it in cell proliferation and tumour formation. It furnishes the factor that is alone wanting to explain the growth of tumours, for, as we have seen, the tissues already possess, in varying degree, the power of cell division and proliferation, and they simply want something to call it into existence. What cells is it likely to influence? Those, naturally, whose proliferating power is greatest. Now special proliferating power may exist under two conditions. First, it may be *natural;* secondly, it may be *acquired*, as I pointed out at the beginning when speaking of certain facts about differentiation. I then showed that loss of differentiation in cells, in other words, loss of functional activity, so that they are no longer of any service in the economy, causes such cells to revert practically to their embryonic state with its unlimited power of proliferation. When dealing with conditions about which we can only theorise, any explanation put forward can be judged in one or two ways. It can be disproved by facts, or it can be strengthened if it is found to be supported by facts on which there is agreement. Let us take a few well-accepted facts and apply the theory.

There is none more generally acknowledged than this: *that long-continued irritation is a predisposing cause of cancer.* What does that mean? It means loss of differentiation and lowering of the functional vitality of the cell, and thereby a greater proliferating power. Again, what fact is more recognised than *that organs where senile changes occur early, as, for instance, the breast and uterus, are especially the seats of cancer.* What is it that happens in these senile changes? There is a physiological atrophy and reversion to a lower type of tissue, so that these organs simply need such a stimulus as I have described to start the commencement of cell proliferation. Further, what fact is there that is more acknowledged than the one that has been brought out Mr. Tatham in his communication to this debate, that when the natural powers of the body are declining, in other words, *when age comes on, cancer manifests itself most?* Recently Dr. Woodhead and others have shown what is the nature of these changes in old age. They are characterised by the loss of the sub-epithelial tissue and by the greater activity of the epithelial cells, these latter showing an increase, with a tendency to dip down and to proliferate. In the same way Cohnheim's inclusions, Ribbert's isolations, and Adami's groups of cells are all liable to be the seat of tumour growth, because their tissue standard is low, and under a stimulus they will begin to proliferate. I removed once a tumour from the parotid region of a young woman, which showed itself when she was about twenty-four years of age. It was a myxo-fibro-chondro-adenoma, and sections of it showed prickly cells scattered therein. No other explanation of such a tumour could be given than that there had been a congenital inclusion (Cohnheim) in connection with the region affected; but why had it not shown itself all the earlier years of her life? As puberty came on, in all probability that fluid of which I have spoken made itself felt, and this tumour which contained the elements of rapid

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proliferation only wanted a stimulus. It received the required stimulus, and accordingly began to grow just previously to her marriage, and when I removed it she had to wean her first child.

There is another point, namely, that of heredity in tumour growths, to which I would like to refer. The view I put forward to you affects not only this but the question of heredity generally. As you know, the theory regarding the latter that has probably received most acceptance is that of the continuous germ plasm (Weissman). I have never been much taken with that theory. I consider that the theory of a secretion from the whole of the tissues meets better the question of general heredity. As regards heredity and tumour formation, my experience at the Glasgow Cancer Hospital is that heredity cannot be put down as a strong factor. I do find this, however, which others also have observed, that there is in the family history of a great many cases of carcinoma evidence of great *longevity*, implying an inheritance of a certain inherent vitality of the tissues, which I consider a factor in the question of the so-called hereditary tendency to tumour growth. Among my hospital patients there was a most interesting case bearing on this point. The patient was an elderly woman, æt. 72, who developed carcinoma of the breast. Her son, who had a severe burn in early life, developed, between thirty and forty years of age, an epithelioma in the scar long before his mother was attacked by carcinoma. He showed the tendency to this disease when young, because in his case it was brought to the surface early by means of the lower differentiated tissue in the scar of the burn, while in the mother it showed itself later in life, only when her tissues had begun to deteriorate and she was advanced in years.

As regards cancer houses and cancer localities, it is a point to which my theory can also apply. If there is one thing which we are agreed upon it is that our surroundings affect our vitality and well-being. If you have an environment which lowers the vitality of the body cells in the sense of lessening their functional activity, you can have coming into play the force connected with the fructifying fluid. It is a curious fact that the statistics in Glasgow of cancer show that out of 1000 certificated deaths there are about 20 from cancer. If you go, however, to the East Coast you have in Dundee 28 per 1000, in Aberdeen 43, and in Edinburgh (only forty miles from Glasgow) you have 49. Is that to be explained on a parasitic theory? I consider that impossible. It is explained on local conditions of environment which affect the dwellers in the towns, interfering with the vitality of the general tissues of the body.

Passing next to carcinoma during pregnancy and to mammary recurrences after long intervals, I would point out that it is well known that when cancer occurs during pregnancy it grows with a rapidity quite out of proportion to what is seen in ordinary cases. It must be that during pregnancy the tissues of the body generally are stimulated to produce material for the service of the tissues. If there is present the fluid such as I have described, it is likely that it will be more in evidence in the condition we are considering, and cancer would flourish more. As regards mammary cancers that recur at long intervals, I had recently a patient who came to me with a small carcinomatous nodule just above the scar left after removal of the breast eleven years previously. Now why should that have been quiescent during all those eleven years? I believe the explanation is that when I removed that breast there was left behind a portion of the gland tissue that had not deteriorated and had not lost its differentiation sufficient to become the seat of cancer. As time went on the tissue came into that condition, and the same influences were brought into play as in the primary growth ten years before, with the result of the nodule in the scar.

My views, then, on the cancer process are as follows:

1. That it can only develop in tissues that have special proliferating power, either natural, as in "Cohnheim's rests," or acquired in one of many possible ways.

2. That the actual exciting cause of the cell proliferation is a secretion from all the tissues of the body and possessed of fructifying powers.

3. That a mutual relationship exists between the character and amount of this secretion and the testes and the ovaries.

4. That another factor in connection with it is the inherent vitality of the tissues themselves.

In this way alone can one satisfactorily explain the disappearance of cancerous tissue under oöphorectomy. It is an important and instructive clinical fact apart altogether from its therapeutical

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value. Such a view of carcinoma is apparently a return to the old spermatic theory. It differs from it, however, in a very important point: it does not imply a conversion of the normal tissue into another kind of tissue. Cancerous tissue is composed of ordinary body cells. The only feature is their intense proliferative power. All that this body secretion does is to furnish the stimulus for proliferation when the condition of the cells allows.

At this point I will read to you a quotation which shows how the lines of thought that come to one are, in reality, common property. The late Dr. Ritchie, whose work on 'Ovarian Physiology and Pathology' is full of interest, makes on the concluding page of it the following suggestive remark: "Perhaps, by the careful study of ovarian growths, our knowledge of the nature of malignancy may some day be signally increased." He also adds: "It is well to bear in mind what Dr. Bright enunciates so well, that the ovaries in their natural structure seem to present a prototype of that involved system of cellular enlargement observed in malignant growths."

In regard to general principles of treatment, from what I have said you will understand that preventive treatment cannot altogether be ignored, and simple tumours should be excised when they exist. I am a firm believer in the local treatment of carcinoma by removal with the knife, and if that is impossible in certain parts of the face caustics may be used. The removal, of course, should be as wide as possible, so as to include any mischief in connection with the growth. In treating limited local recurrences excision by the knife should be practised. In one or two cases that I have under the treatment of the Röntgen rays, it would appear that this means may be found to be an important element in their removal. It should always be remembered that however carefully the knife is used new tissues are opened up, and that leads to an activity that may do harm. In rodent ulcers unsuitable for excision I think the X rays are of value. In those cases where you have no visceral complications with inoperable mammary cancer, I have no hesitation in recommending oöphorectomy and thyroid treatment. It has undoubtedly proved of use in many cases. In the first case that I operated on all the cancerous tissue disappeared. The patient was a young woman æt. 33, suffering from a recurrent inoperable mammary cancer, and she lived for four years after the operation, dying from a secondary growth in the spinal cord, probably caused by a cell which, I think, had possibly hived off at an early period. As regards thyroid extract, I have given as much as 250 grains of it in the day without any great physiological effect; but I have come to the conclusion that from five, ten, to fifteen grains per day is as much as is necessary, and produces all the metabolic changes that are useful in this disease. In other inoperable cases, such as cancer of the rectum, the practice of colostomy will prolong life, for, to put it briefly, the means by which the cancer kills the patient are to be provided against as far as possible.

In conclusion, I would say that working at cancer is really very disappointing work. All that one can say is:

> "The reward is in the doing,
> And the rapture of pursuing
> Is the prize;"

but by holding debates and meetings such as the present, individual effort is encouraged, and collective work is stimulated. In this way only will true success come, and it is by such means that we may hope to wipe away the present reproach that cancer is to our professional skill.

The Relation of the Bacillus of Diphtheria in Man to the Bacillus of Bird Diphtheria. By A. M. Maksutoff.—The bacillus of bird diphtheria is classed by consensus of opinion among the pseudo-diphtheria bacilli. A study of the bacilli isolated by culture from hens affected with diphtheria shows that these germs present considerable differences as compared to those of human diphtheria. The author concludes, however, as a result of his investigations, that the bacillus of chicken diphtheria, while not belonging to the same species of germs as that of human diphtheria, certainly is more closely related to the latter than to the pseudo-diphtheria bacilli. He has also found that Neisser's method of staining does not appear characteristic for the Klebs-Löffler bacillus, as was formerly supposed, for the bacilli of chicken diphtheria stained with this method.—*New York Med. Journ.*, April 26th, 1902.

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ON THE TREATMENT OF INVETERATE RETROFLEXION OF THE UTERUS.

A Clinical Lecture delivered at the Medical Graduates' College and Polyclinic on May 21st, 1902.

By J. BLAND-SUTTON, F.R.C.S.,

Surgeon to the Chelsea Hospital for Women; Assistant Surgeon, Middlesex Hospital.

PESSARIES, like trusses, have their proper sphere of usefulness, but modern surgery has considerably curtailed the employment of trusses, and I will endeavour to show that in the treatment of retroflexion of the uterus pessaries are useless, and in very many instances harmful. In making this statement I fully realise that I am speaking as a general surgeon attached to a large general hospital, but I am also surgeon to the Chelsea Hospital for Women, where a large amount of uterine surgery comes under my personal care.

For some years it has been very clearly forced upon me that in cases where retroflexion of the uterus is treated by pessaries, and these "foreign bodies" are tolerated, they are usually unnecessary, whereas those patients who seem to need some kind of support find pessaries intolerable.

My capacity for appreciating mechanics, either theoretical or practical, is not great, and certainly the lever action supposed to be exerted by a pessary, as described by its ardent advocates, I never could understand; but I am dominated by a very active scepticism in most things, and certainly my doubts concerning the utility of pessaries in maintaining the rectitude of the uterus were aroused in a very natural way. Many women have come under my care after being treated by experienced obstetric physicians for supposed retroflexion of the uterus. Having reason to doubt the diagnosis, I have recommended them to submit to operation, and have had the satisfaction of removing small ovarian cysts or dermoids, chronically inflamed

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Fallopian tubes, or small pedunculated fibroids, which had been mistaken for a retroflexed fundus of the uterus. I am bold enough to state that there are few pelvic conditions concerning which so many mistakes are made in diagnosis, as retroflexion of the uterus. Quite apart from any error of diagnosis, let me tell how I submitted the supposed efficacy of the Hodge and the ring pessary to a test. I advised a patient with severe and inveterate retroflexion of the uterus to submit to hysteropexy. On the day of the operation I straightened the uterus with a sound, and introduced the Hodge she had been wearing for many months. About four hours later I operated, and on exposing the pelvic organs through an abdominal incision the uterus was found acutely retroflexed, and the only effect the pessary exercised was to push the whole organ higher in the pelvis than usual. On a subsequent occasion I carried out the same test with the rubber ring pessary, and with the same result.

In my early days, when I used to haunt the *post-mortem* room, I was very sceptical in regard to backward displacement of the uterus producing troublesome symptoms. Indeed, one occasionally found the uterus so acutely retroflexed that the fundus rested upon the floor of the pelvis; yet there would be nothing in the clinical record of the case suggesting that the displacement had produced any inconvenience. Some of the most marked specimens were found in girls of seventeen to eighteen years of age. There are, however, conditions in which retroflexion of the uterus is a painful and distressing condition, but this depends on the character of the displacement, and particularly on the position the ovaries occupy in regard to the displaced body of the uterus.

In the observations which follow no attempt will be made to distinguish between retroflexion, with or without retroversion, or to differentiate between retroflexion affecting the body of the uterus or its cervix, but the term retroflexion will be used in a general sense to signify all backward displacements of the uterus, but have no reference to prolapse of this organ.

Without entering too minutely into the question of the normal position of the uterus, it may be stated that this organ is so situated in the pelvis that its anterior surface is somewhat concave where it comes in contact with the bladder, and that in a well-developed woman, especially one who has borne children, the summit of the uterus is on a level with the upper border of the pubes. The ovaries lie on a level with the pelvic brim. When the uterus becomes acutely retroflexed the ovaries may remain practically undisturbed in their normal position. This commonly, but not invariably, happens in sterile women, but in very many patients the ovaries and tubes are much displaced, and their relation to the fundus of the uterus is of great importance. It is not uncommon to find the uterus acutely retroflexed, and one or both ovaries with the corresponding section of the Fallopian tubes lying beneath the uterus.

This condition, though by no means confined to patients who have had children, is more frequent in them than in sterile women. This is due to the fact that pregnancy stretches all the structures connected with the uterus, and when frequently repeated leaves them very lax indeed.

When one or both ovaries lie on the bottom of the recto-vaginal fossa with the body of the uterus resting upon them they are sources of pain, and the rolling movement which the uterus exercises on an ovary in this position will often cause it to be covered with a thick patch of fibrous tissue, resembling the familiar white patch on the anterior surface of the heart. When one or both ovaries are entrapped in this way, and a pessary is introduced, it occasionally happens that the posterior limb of the pessary will work into such a position that it comes to lie between the fundus of the uterus and the ovaries. In such a case on examining the patient with the pessary in position the ovaries can be felt low in the recto-vaginal fossa below the pessary. The pressure of the pessary produces great pain and will cause the ovaries to become œdematous, a fact sometimes demonstrable in the course of an operation.

Although in a very large number of cases ovaries are dragged from their normal position secondary to the displacement of the uterus, it must not be forgotten that the converse of this is often true, namely, that an ovary enlarged by a simple cyst, a dermoid, or from any cause, and though the gland may not exceed the dimensions of a walnut, will descend from the brim of the pelvis to the floor of the recto-vaginal fossa, and by its weight drag the uterus backward. Any smooth small body of this kind lying at the back of the uterus in a virgin,

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whose narrow vagina does not facilitate examination, often resembles a retroflexed fundus; as the uterus itself is, in such circumstances, often drawn backwards the employment of a sound assists in the perpetration of an erroneous diagnosis. In such cases the only sure way of avoiding error is to examine the patient during anæsthesia.

The variety of backward displacement of the uterus which most frequently calls for interference, and which is most productive of distress, occurs in women who have borne children, for the uterus softened by repeated pregnancies will, when it becomes displaced backwards, so accurately adjust itself to the recto-vaginal fossa that when extracted from the grip of the utero-sacral ligaments its posterior surface will present an exact cast of this recess. A uterus moulded in the recto-vaginal fossa in this way is often very troublesome, for, apart from producing discomfort, it will press on the lower segment of the rectum and induce mechanical constipation.

There is a peculiar condition sometimes associated with a softened and impacted uterus: when its fundus accurately fits the recto-vaginal pouch, fluid (lymph) sometimes exudes between the uterus and the floor of the fossa, and as this exudation cannot escape, the tension it produces may not only cause discomfort, but debar the patient from active duties. When operations are undertaken in such conditions the fluid sponged out of the recto-vaginal fossa may measure two or more ounces. It is usually of a light pink, and quickly coagulates.

In a small proportion of cases the body of the uterus is fixed sometimes very firmly to the floor of the pelvis. A careful inquiry into the clinical history of these cases usually affords evidence of an old septic endometritis, a sequel, either of a confinement, or of a miscarriage.

Another important cause of retroflexion is a small fibroid in the posterior wall of the uterus, but I do not propose to speak of this complication on the present occasion, although it is a question of great interest and importance.

There is no subject in practical gynæcology which has been the subject of so much satire in the profession and out of it as the treatment of retroflexion of the uterus. On well-worn ground such as this I do not propose to stray, but my object in this lecture is to urge that there are conditions in which a retroflexed uterus is a source of suffering and demands active treatment, and that the most satisfactory manner of affording relief to these severe and inveterate cases is by means of the operation known as hysteropexy, whereby the fundus of the uterus is secured by means of sutures to the anterior abdominal wall.

One of the greatest objections urged against this operation is that it fixes the uterus in an abnormal position. This is not quite true, for as I pointed out in the early part of the lecture, the fundus of the uterus in a woman who has had children is on a level with the upper border of the symphysis pubis, and on bimanual examination this part of the uterus can, in a thin woman, be easily made to come in contact with the abdominal wall at this point.

There is a question, and a very pertinent question, almost invariably asked by those who witness a hysteropexy for the first time, and that is, the way in which the uterus behaves in the event of a subsequent pregnancy.

In the early stages of this operation, when it was the custom to fix the uterus very securely by a number of strong sutures to the anterior abdominal wall, some grievous results were reported: miscarriages were said to be more frequent, obstructed labours and even rupture of the uterus were reported as sequelæ. From my own experience I cannot state anything regarding such disasters. I select the patients for this operation with very great care, and limit the operation to the very bad and inveterate cases, and I always keep constantly in mind this very important fact: *Pregnancy is the best cure for a retroflexed uterus*, and it is only after a very careful consideration of the improbability of this natural mode of cure coming about that I advise hysteropexy, and in the majority of patients in the table furnished on page 84 there had been in the majority a period of sterility, in those that were married, varying from three to ten years. On the other hand, in two or three cases where the subsequent history of the patients came under my notice the hysteropexy seemed to have facilitated fertilisation, for the patients again became mothers and their pregnancies had happy terminations.

There is one kind of patient in whom retroflexion of the uterus is often accompanied with a manifestation of extreme distress, and in whom hysteropexy is strongly contra-indicated, and that is in

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"alcoholics." Too much care cannot be taken, in selecting patients for this operation, to exclude women where there is a suspicion of this vice.

The accompanying table represents a consecutive series of cases under my care in the Chelsea Hospital for Women in which hysteropexy was performed. The table, which covers a period of five years, was drawn up by my colleague, Dr. Victor Bonney. It is a feature in this series of cases that the married women, with few exceptions, belong to two decades, twenty-five to thirty-five, and thirty-five to forty-five.

Case 40 in the list is the youngest, and perhaps the most remarkable of all. Before she came under my care her right ovary had been removed, and hysteropexy performed by another surgeon, but the retroflexion returned. I operated on her some months later, and found the left ovary full of cysts, but being very unwilling to remove it, I punctured the ovary very freely and stitched the uterus to the abdominal wall. In spite of the unsatisfactory character of the ovary this patient quickly conceived, and her pregnancy had a very happy issue.

Hysteropexy.—The patient is prepared for this operation in the following way:—As it is never necessary to perform hysteropexy as an operation of emergency, the patient may have the advantage of thirty-six or forty-eight hours' rest in bed. This allows time to have the bowels thoroughly evacuated, and affords every opportunity for the nurse to make the skin of the abdomen as antiseptic as soap and water, with the subsequent use of perchloride of mercury solution (one in 2000) or some similar efficacious preparation, will make it. For the operation the Trendelenburg position is indispensable. The following instruments are sufficient:—a scalpel; six hæmostatic forceps; dissecting forceps; a few needles, curved and straight; silk of various sizes; a few strands of silkworm gut; six gauze dabs; a glass catheter. All the instruments are boiled for fifteen minutes immediately before the operation. The dabs are boiled for one hour, and the silk and silkworm gut for thirty minutes. Before the patient is placed on the table, the nurse empties the bladder with a sterilised glass catheter. The anæsthetic varies according to the condition of the patient, and in this matter I always leave the choice of the reagent to the anæsthetist.

Table of Fifty Consecutive Cases of Hysteropexy performed between November, 1896, and November, 1901, for Inveterate Retroflexion of the Uterus at the Chelsea Hospital for Women.

No.	Age.	Social state, No. of pregnancies.	Complications.
1	47	M. 1	
2	24	M. 1	
3	40	M. 2	
4	42	M. 3	
5	40	M. 1	
6	33	M. 3	One ovary removed.
7	38	M. 3	
8	35	M. 0	
9	48	M. 4	
10	55	M. 0	
11	44	M. 3	
12	27	M. 2	Right ovary removed.
13	24	S. 1	
14	34	M. 2	
15	30	M. 2	Left ovary removed.
16	32	S. 0	
17	29	M. 2	
18	37	M. 0	
19	28	M. 1	
20	32	S. 0	
21	43	S. 0	
22	30	M. 2	
23	25	S. 0	
24	34	M. 5	Both ovaries removed.
25	25	M. 3	
26	32	M. 0	Right ovary removed.
27	23	S. 0	
28	23	S. 0	Left ovary removed.
29	48	S. 0	
30	35	M. 0	
31	30	M. 1	Bilateral hydrosalpinx removed.
32	27	M. 0	
33	43	M. 1	
34	41	M. 6	
35	27	S. 0	An encysted collection of fluid under the incarcerated fundus.
36	32	M. 1	
37	30	M. 3	
38	30	S. 0	
39	33	M. 0	
40	21	M. 1	
41	43	M. 1	
42	25	M. 5	
43	37	M. 2	
44	27	M. 2	
45	27	M. 2	
46	42	M. 2	Left ovary removed.
47	32	M. 7	
48	37	M. 2	
49	33	M. 1	Left ovary removed.
50	37	M. 3	

The abdomen is then opened in the middle line, as for ovariotomy. The doubled-up uterus is then sought for in the pelvis, and, if it be free from adhesions, is at once straightened and brought into

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position. The operator then ascertains the condition of the ovaries and tubes, and, if he is satisfied with them, he proceeds to secure the fundus of the uterus to the abdominal wall.

Should an ovary contain small cysts, these should be punctured with the point of the scalpel, and the fluid squeezed out. An enlarged and prolapsed ovary may require removal, and in a certain proportion of cases an ovary may be converted into a dermoid the size of a bantam's egg, and demand removal.

Occasionally the retroflexion is due to a fibroid in the posterior wall of the uterus; in such a case the tumour should be enucleated and the capsule closed with sutures.

The fundus of the uterus may be so fixed with adhesions to the floor of the recto-vaginal fossa as to require very great care in raising it, especially if the adhesions bind it closely to the rectum. In some cases, too, a collection of peritoneal fluid (lymph) will be found below the displaced body of the uterus and shut in by it, so that when the fundus is raised this fluid will escape, and for a moment will lead the surgeon to think that he has burst a thin-walled cyst.

When the operator has satisfied himself that the pelvic conditions are in order, he passes a curved needle, armed with a strand of silk-worm gut, through the aponeurosis and adjacent peritoneum, on one edge of the wound, and carries it through the fundus of the uterus, and then brings it out through peritoneum and aponeurosis on the opposite edge of the incision; when this suture is tightened it brings the edges of the incision and the fundus of the uterus in close contact. A second suture is passed in the same way for additional security. All fluid in the pelvic *cul-de-sac* is now carefully sponged out, the forceps and dabs counted to insure that none are left in the belly, and the incision is secured in the following way:—The edges of the peritoneum are united with fine silk; the fascial sheaths of the rectus muscles are brought together with interrupted sutures of silkworm gut, and the skin sutured with thin silk.

A light dressing of sterilised gauze and some cotton wool, or preferably Gamgee tissue, is held in place by a many-tail of flannel, and the patient returned to bed.

The after-treatment calls for no comment, for convalescence is, as a rule, rapid and uneventful. It is wise to keep the patient resting in bed three weeks. On three or four occasions it has happened that a stitch abscess formed over the retaining sutures, and necessitated their removal, and I have found that this is likely to arise when hysteropexy is performed on the large soft uteri of multiparæ. This is due to the fact that the needle penetrates the uterus too deeply, and the sutures are infected from the uterine cavity. Knowledge of the cause has prevented this complication in subsequent cases.

The operation is one of the simplest that can be performed on any of the abdominal viscera; in an uncomplicated case it can be carried out in a few minutes, and if the aseptic method is followed with proper care in all its details the risks are extremely small, and the post-operative course of the case is painless. I do not think it worth while to discuss the various methods which have been advocated for suspending a misplaced uterus. Most surgeons who have devoted their attention to this operation prefer the abdominal methods, which I have just described. The chief difficulties seem rather to have been in the choice of material for suture. In my early operations I employed carefully sterilised silk, then for a brief period I used catgut, but this material I discarded absolutely in 1896, and have never used it in any operation since. It is unreliable and difficult to sterilise, and I feel that more lives are annually destroyed from the use of badly prepared catgut in the name of surgery than from rope in the name of justice. For several years I have exclusively employed silkworm gut; it is reliable, and does not deteriorate after boiling half an hour in the steriliser; it does not absorb for many years. I have found a piece of silkworm gut unchanged after remaining buried in a sound cicatrix four years.

Gentlemen, let me assure you that this lecture is in no sense a diatribe upon pessaries; abler and older men than myself have "poured out the vials of their wrath" upon these curious inventions, and it is as useless to deal with this subject with contemptuous raillery as to flog the sea for roaring when it is irritated by a gale of wind; therefore let me set before you some facts which will reveal how deeply rooted the employment of pessaries is in the treatment of uterine misplacements, real and imaginary.

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I obtained from a London maker the statement that in a year he supplies about 10,000 rubber rings and globular pessaries, and about 3000 Hodges (as they are familiarly called) made of vulcanite.

Another London maker supplied in one month 286 dozen; the average for the year is a little below this, and runs to about 250 dozen rings, Hodge and Greenhalgh's patterns. This in a year would amount to 36,000. To these statistics each of these makers add the following significant remarks:—"Vast quantities of pessaries are imported from Germany and other countries." The numbers already quoted do not include metal pessaries. As there are several makers of rubber and vulcanite pessaries in London, these few facts may help us to obtain some faint notion of the extent to which faith in pessaries abounds.

In conclusion, it may be observed that as every man considers himself a judge of horses, so every practitioner has his own view on the utility or the reverse of a pessary, and nearly every gynæcologist has his own pet notions concerning the shape of pessaries or the material of which they are made. For my own part I frankly confess that there is only one place for pessaries designed to cure retroflexion of the uterus, and that is the fire.

How should Organotherapy be Practised? By Dr. G. Zanoni.—The safest and quickest way of employing organotherapeutic substances is by using the glycerine extracts hypodermically. In order to judge with certainty the therapeutic effect of these preparations, one must be sure that the extract has been properly prepared. The colour of the extract must be observed, for they are not completely colourless, but have tints which are peculiar to each particular extract, so that they may be recognised by this characteristic alone. An important point is the transparency of the extract, and it must be noted that when an extract becomes turbid it loses its activity. The strength of the extract may be tested in a general way by measuring the amount of albuminous substances it contains, by precipitating the latter by means of absolute alcohol, or by the ordinary reactions for albumin.—*N. Y. Med. Journ.*, May 3, 1902.

ON THE PREVENTION AND TREATMENT OF MIDDLE EAR SUPPURATION.*

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At your secretary's request I propose to make a few remarks on some points connected with middle ear suppuration, because my experience is similar to that of most surgeons specially interested in otology, that its importance in frequently producing fatal results, and in an even larger percentage of cases great depreciation of the general health, is not sufficiently impressed upon those unfortunately suffering from it.

The most common statement made by parents when blamed for, perhaps, many years' neglect of middle ear suppuration is that they were advised to leave it alone as the patient would outgrow it, an opinion entirely contrary to the facts of general pathology, and indicating a very pessimistic opinion of the progress made in recent years by scientific otology.

One of the pioneers of aural surgery in this kingdom, Sir William Wilde, is often quoted as saying, "When a discharge from the ear exists, we can never tell how, when, or where it will end, or to what it may lead." Statistics † as to the frequency of middle ear suppuration in childhood are unattainable, and for our purpose unnecessary, for its frequency is common knowledge.

One of the chief evils which frequently comes under notice is the quiescence during childhood and adolescence of otitis media after an acute onset, little watery discharge alone remaining, which in later young adult life is most intractable, and frequently gives rise to fatal complications. That these fatal complications are not a myth of the aurist is shown by Pitt's statistics ("Goulstonian Lectures," 'British Medical Journal,' vol. i, p.

* Delivered before the South London Branch of the British Medical Association at the Evelina Hospital, February 27th, 1902.

† Mr. A. H. Cheatle has since (Otological Society's Meeting, April 14th) reported that of 1000 board school children examined in the Hanwell schools, 335 were suffering, or had suffered, from middle ear suppuration.

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643, 1890), which show that out of 9000 autopsies, middle ear suppuration was the known cause of death in one in 158, and, as Körner ('Otitische Erkrankungen des Hirns, etc.') points out, many are doubtless set down as convulsions, pyæmia, and other unclassified diseases.

The frequency of chronic disease of the temporal bone spreading from the lining membrane of the middle ear has been indelibly fixed upon my mind by a three years' examination of all available bodies in the Guy's Hospital dissecting room, and I can only say that here healthy middle ears are the exception rather than the rule; of the specimens I am showing to-day obtained from this source, one shows extension backwards of disease by a large cavity laying bare the dura mater into the posterior fossa, and several others show extensive caries of the osseous middle ear and adjacent cavities.

Complications are, as we know, most common between eleven and thirty, between which years 58 per cent. of all complications occur, which, according to Körner, probably average 4·5 per cent. of all causes of death recorded during this period of life.

To the importance of these facts the soundest of the life insurance institutions are gradually awakening, and a specialist's opinion is now often asked, for it will be granted that in no other specialist's realm do the companies run such risks in the earlier years of the insured's life.

These last considerations do not so much concern the general practitioner, though it is really on him that the whole matter depends, for if in his hands does not lie the operative treatment of chronic otorrhœa (probably its only cure) most certainly in his hands rests the prophylaxis of this terrible disease, and it is in the future by preventive measures that the number of mortalities must be diminished. The well-known common causes of acute mischief are the specific fevers, of which by far the most troublesome in treatment and frequent in intra-cranial complications is influenza. Further, it is always well to bear in mind, in a case of otorrhœa commencing painlessly, the possibility of tubercle as a cause. I would suggest it as most common in extreme infancy, when its association as causative factor in tuberculous meningitis is well known. There is one point in this connection which is, I think, of great diagnostic value, namely, a gradual enlargement of the infra-mastoid lymphatic glands in a child suffering from chronic otorrhœa, indicating the possibility of the whole process being tuberculous, as was the case in the specimen which I now show you.

Apart from specific fevers, we commonly find in a severe cold the causation of most cases of middle ear suppuration, and this is especially so in those cases in which the common history of the mother is, "He has often had it before, every cold flies to his throat, and then his ears begin to ache."

The line of treatment is almost thrust into our hands; we cannot too strongly insist that earache and otorrhœa are not normal physiological processes, innately connected with dentition or puberty, but are grave pathological states, and it is with the prophylactic treatment of middle ear inflammation in this stage that I specially wish to trouble you to-day. When we meet the severe intermittent earache and deafness of sudden onset in childhood, so suggestive of acute otitis media, the first condition to investigate is the state of the tympanic membrane, for it is of the greatest importance to recognise the injected opaque, often bulging drum of this acute stage, as failure to recognise by direct inspection the warnings of such acute inflammation is so commonly followed by the very result which, of all others in inflammation elsewhere in the body, we wish to avoid, namely, suppuration, which process is here followed, at a more or less prolonged interval of pain and fever, by perforation.

Of all prophylactic measures which is gradually becoming estimated at its true value in the prevention of suppuration, undoubtedly paracentesis is of most importance. In many cases leeching, fomentations, instillation of oil containing 2 per cent. of acetate of morphia, narcotics to produce sleep, will in twenty-four to thirty-six hours cause a cessation of pain and a great improvement of the general condition. I am myself in favour in the painful stage of hourly repeated injections of carbolic lotion 1 in 40, after a thorough cleansing of the meatus with soap and water, turpentine and ether, because if the lotion be used as hot as possible the pain is much relieved, and, further, its use paves the way, by attempting to render the meatus aseptic, for paracentesis, when necessary, of which the only risk is a further infection of the middle ear from outside

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by the paracentesis needle, speculum, or the operator's fingers.

I have said that local medication should at first be given a trial, and it is very difficult to establish a time limit, but I think that if severe pain resists treatment for twenty-four hours, and on examination the redness and swelling of the membrane has increased or remains, especially if the most bulging part presents a yellowish green appearance, indicating a tendency to perforation, then paracentesis must be performed. The most convenient paracentesis needle to my mind is bayonet-shaped, the point of which, guided by direct inspection with a good light, is sharply yet carefully thrust through the posterior-inferior segment of the drum, care being taken to avoid the malleus, descending process of the incus, and the inner wall of the middle ear. No anæsthetic is necessary, local antiseptic measures must be as carefully as possible carried out, and it is better to err on the side of doing it when not absolutely needful than neglecting it when necessary, for in a favourable case it will generally be found that sanious, and later sero-mucous discharge persist for a few days and within a week, under local antiseptic treatment and the daily use of Politzer's bag, that the operation wound has healed, and the hearing is gradually returning to the normal.

It is unnecessary to remark that such a favourable result is rarely seen in acute ear disease allowed to continue to suppuration and perforation, and it is my fixed opinion that by this means alone, more judiciously and generally used, many cases of chronic suppuration would be avoided.

After local examination of both membranes it is in every case most important to ascertain the condition of the pharynx, either by the post-nasal mirror or with the finger, to estimate and treat, if present, acute pharyngitis, and above all to detect the presence of adenoids.

Much has been written in recent years about the non-operative treatment of adenoids, but I venture to think that no one would be so rash as to advise that adenoids be left alone with a history of recurrent attacks of severe earache or when discovered during an attack of otitis media. Of course no operation for their removal should be undertaken during an acute attack of middle ear inflammation, but given a definite local lymphoid hypertrophy, and not a transient engorgement of the normal pharyngeal tonsil, no time must be lost in its removal after the cessation of the attack. I have had unique opportunities of watching and comparing the results of non-operative and operative measures, and can only say that in the treatment of acute inflammation, before or after suppuration, removal of adenoids has been for me, as for nearly all aural surgeons, followed by the best possible results.

It is my experience that in childhood bilateral chronic middle ear suppuration is, in the majority of cases, associated with adenoids, in many of which persistent antiseptic treatment has, without their removal, been of no avail.

If first seen in the acute stage of suppuration or in cases where attempts to prevent suppuration have failed, it is well to enlarge the perforation if small and discharge is apparently retained, and, if circumstances permit, daily cleansing and instillation of 33 per cent. alcohol drops and packing with a thin strip of sterilised gauze is the best line of treatment to adopt; but it is obvious that this, which requires daily observation, cannot be always carried out.

During acute suppuration complications are extremely rare, Grunert (Schwartze, 'Handbuch der Ohrenheilkunde'), 11,849, found 91 per cent. of brain abscesses caused by chronic, and only 9 per cent. by acute middle ear suppuration; and Mac Ewen 'Pyogenic Diseases of Brain,' regards acute otitis media as even more rarely still causing intra-cranial complications.

After an arbitrary period of some six weeks the suppuration is called chronic, and even in this stage a large proportion of cases may be cured by removal of adenoids, inflation with Politzer's bag, and persistent syringing with carbolic lotion 1 in 60, or formalin 1 in 500; a useful adjunct here is the use of absolute alcohol, 33 per cent. instilled for ten minutes, or of hydrogen peroxide 10 to 15 vols. for ten minutes before syringing, to disintegrate the necrotic tissues, and in the same way I am trying at present an artificial gastric juice which has much to recommend it in breaking up the results of tissue necrosis.

It is impossible here to give the exact indications for one or other of these lines of treatment, and it will be seen that my recommendations are towards the wet as opposed to the dry method of treatment. In spite of all this it will be found that

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certain cases will, even after six months' careful and continuous treatment on these lines and removal of granulations should they spring up, still continue to discharge, and, in fact, be very little altered.

In these cases it will generally be found that there are evidences of bone disease, either as discovered by a probe introduced through the perforation into the middle ear or suggested by the situation of the perforation, which is usually either in Shrapnell's membrane or in the posterior-superior segment involving the posterior malleal fold, and with these perforations it is common to find either the descending process of the incus or the head of the malleus eroded as shown by these specimens. Such conditions may be also suspected by the presence of a profuse, watery, evil-smelling discharge, in which many epithelial elements and fragments of bone *débris* are to be found under the microscope. As the carious process spreads to the parietal walls of the antro-tympanic cavity, the ulcerated mucosa throws off large masses of disintegrating cells, of which the fluid parts drain away in the watery discharge, leaving behind the epithelial elements in an onion-like mass called cholesteatoma. Such cases are characterised by frequent recurrence of polypi, even after careful removal with the snare. When this stage is reached we must be always on the watch for evil symptoms, of which the most useful, as indicating extension of the various processes and possible infection of the outer wall of the dura mater, are headache, giddiness, somnolence, facial paralysis, and vomiting.

In the present day it is quite unjustifiable to wait for such signs without undertaking operative measures, the first of which, in cases where there is no reason to suspect extension of caries from the ossicles to the parietal walls of the middle ear, should be ossiculectomy or excision of the membrane, malleus, and incus, and removal of the outer attic wall (*vide* paper by author, 'Guy's Hospital Reports, 1897').

In cases of cholesteatoma, or where there are other indications of affections of the bony walls of the middle ear, the radical mastoid operation must be undertaken after the failure of six months' antiseptic treatment with or without removal of ossicles. The steps of this operation it is unnecessary to mention here, its results can be seen by the children I am bringing before you to-day. I should like, however, to conclude by briefly summarising its indications:

1. Failure of six months' antiseptic treatment with a clear pharynx.
2. Special perforations, *e. g.* in Shrapnell's membrane.
3. Evidences of caries with probe.
4. Warning symptoms, headache, facial paralysis, etc.
5. Tuberculous disease of temporal bone.
6. Acute exacerbation of disease in mastoid with headache, pyrexia, pain on pressure over mastoid, œdema, swelling behind ear in chronic cases.
7. Onset of intra-cranial complications.

And I would like to suggest that until all these measures have been efficiently tried no cases of chronic otorrhœa must be labelled as incurable, for in my experience we have certainly arrived at such a point that only an extremely small percentage if any of such cases can be truly placed in this category.

Hypnotic Action of Apomorphine in Alcoholism. Coleman, W., and Polk, J. M. 'American Medicine,' vol. iii, No. 10, p. 381, March 8th, 1902.—Attention was recently called to the value of apomorphine as a hypnotic in cases of alcoholism. The writers have taken the opportunity to test extensively the use of this remedy, having used it in 300 cases admitted to the Bellevue Hospital. To act as a sedative, apomorphine should be administered hypodermically. In thirteen cases it was given by the mouth, and only two of these patients slept. Of seventy-four cases, it acted as a hypnotic in fifty. Nearly one half of the patients vomited. The dose cannot be definitely stated. It is best to begin with one-thirtieth of a grain, or less, and repeat this in a short time. If vomiting occurs, the drug should not be repeated until several hours have passed. The drug should be avoided in patients who are weak. The hypnotic action lasts only a few hours, and when the patient awakens his condition is practically unchanged, except in "ordinary drunks." The best results are obtained from apomorphine when it is followed in a few hours by a recognised hypnotic, such as choral, bromide, etc.—*Medicine*, May, 1902.

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DEBATE

ON

CANCER: ITS ORIGIN, NATURE, AND GENERAL PRINCIPLES OF TREATMENT.

Held under the auspices of the Chelsea Clinical Society, at the Jenner Institute of Preventive Medicine, March 11th, 18th, and 25th, 1902.

Dr. CHARLES MORRIS, C.V.O., President, in the Chair.

SIR WILLIAM MITCHELL BANKS.—Mr. President and Gentlemen, I shall confine my remarks to-night very definitely to some four separate points in this great question of cancer. I venture to speak not from statistics but from an experience lasting now some thirty-five years, because I have found that, after all, statistics are very dangerous things to manipulate, and can be worked about in various ways, so that I know of no condition which more requires a man to go into his upper chamber and say to himself that he must do honestly, than when he is making statistical tables. There is a good deal of what we may call impressionism, which finally settles down on a man's mind after he has seen a great many things, and I may say that, in the remarks which I have to make, mine will be more of a case of impressionism than of statistics. It is about two years since I gave, in the Lettsomian Lectures, a general review of the existing state of our knowledge of cancer up to that date, and more especially the one form of cancer in which I am interested—cancer of the breast. Two years, then, having elapsed, have we learnt anything more? I am sorry to say that nothing of any moment has occurred to increase our knowledge of the subject during these two years; we are pretty much where we stood, except possibly in one matter, namely the question as to whether electricity is likely to act in a curative manner or not. The points which I should like to say a little about are, first of all with reference to the increase of cancer, and then I should like to mention what my notion of the causation of the disease is, partially at any rate. Then there is the great question of what we are to make out of the so-called cancer parasite, and finally there is the question, Is this an infectious disease, or is it not? Beginning, then, with the point as to the increase of cancer, I think it is now universally admitted that in all civilised countries and among all civilised peoples there has been a very notable, clear, and distinct increase in the amount of cancerous disease, especially arising during the lifetime of men now alive. The last defence of the opposite view was probably made by Dr. Arthur Newsholme in April, 1899, in what was a very valuable number of the 'Practitioner,' which many of you will remember was called "The Cancer Number" of the 'Practitioner.' It contained admirable articles, which will put any one into complete possession of all that was known at that time. Dr. Arthur Newsholme made a stand against this view, and he argued that the so-called increase in cancer was more apparent than real, because he thought that improved diagnosis had made our returns of death certificates much more accurate than formerly, and he believed that necropsies, notably in hospitals, were much more frequent than previously. I do not agree with him in any one single point, and I think the view is now no longer held. The great mass of statistics accumulated during the past two years have been too much for him. And from thirty-five years of pretty strenuous surgical work my knowledge and recollection is that I see a great many more cases of cancer, not in my own practice only, but in everybody else's, than I used to do, and what is even more distressing, I see it beginning to attack younger persons than formerly. Cases of cancer occurring at between twenty and thirty years of age, at which age it was formerly thought to be as unreasonable to expect it as between ten and fifteen, are now not at all unknown; of course I mean carcinoma. Now, if a person comes to you with what is supposed to be cancer of the breast at thirty years of age, I am sorry to say there is no reason why she should not have cancer. Well, there is one very curious point which has been established by statistics, and which I have taken notice of before, and that is that they seem distinctly to show that whilst there is an increase—and a notable increase, in both sexes, that in the case of the male the increase has been greater than that in the female. The point, in short, is that the male is now suffering from cancer in a greater degree proportionately to the female than he used to do. I venture to bring this in for an argument presently in connection

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with another point. The next point I want to allude to is, Have we anything we can positively advance as a great broad general reason for cancer? Of course we have the statistics of Mr. Haviland, perhaps some of the most remarkable and brilliant pieces of statistical work that ever were done,—certainly in this country. The labours of a lifetime have been devoted by Mr. Haviland to the considering of cancer in connection with its relations to geographical situations. You all know, no doubt, that his general view is that cancer is most prevalent in the neighbourhood of rivers which periodically overflow;—low, damp situations apt to be flooded by water from rivers or other sources, and particularly having connection with a clay soil; while the parts where there is usually less cancer are the high districts, and notably those connected with the chalk lands. The latter districts were those in which cancer was less deadly. Well, I think there is no doubt that a good deal must be credited to Mr. Haviland for his labours. But he himself fully admits that he cannot explain it. He says, "I do not profess to know how it comes about, but the collection of my figures goes to show that low-lying periodically flooded areas, with a clay basis, are the localities most favoured by cancer."

Now as to other reasons: they are many. I have gradually formed one of my own; it is not mine specially, and I suspect it is the view of many people. But my attention was first directed in this direction by taking a patient to the late Sir James Paget with cancer of the breast. I remember him saying to me—and it is a great many years ago—"We must try and starve her." His belief was that people who had cancer ought to eat but very little. I often thought of this, and as I came to think more and more about it, and more information reached me, I have arrived at the idea that possibly one of the factors in the production of cancer is the great increase in the amount of food, and notably meat, which is now consumed by all civilised people. How long has this question of the increase in cancer been noticed? Probably not more than thirty or forty years. If you read the writings of 1850 you will not find anybody taking any notice of it at all. In no periodical will you find a statement by a surgeon of that day that cancer was increasing. It is only during the last generation, which I think is thirty-three years, that this question has come more to the fore. Well, if that is so, what are the notable changes in the life of civilised mankind that have occurred? Can we put many factors out by the process of exclusion? For instance, is drunkenness a cause? I am certain that drunkenness has nothing to do with cancer. Take women with cancer of the breast. It is the form of cancer which is most familiar. I have seen very many of these, and I can certainly say that I would never for a moment accuse them of being a body of persons given to drink, but probably the very opposite. I have not seen any evidence whatever that drink has anything to do with cancer. The next point is as to syphilis. Has that become more widely and more generally spread amongst civilised people, and is consequently a cause? I see nothing in syphilis to account for it at all. Again, I will take the question of the women with cancer of the breast, and I am fortified in this view by the opinion of Mr. Roger Williams, whose collections of statistics upon all matters connected with cancerous disease are quite remarkable. He, for instance, inquired carefully into the cases of some 160 or more women with cancer of the breast, and he saw no reason at all to think that syphilis was a cause of cancer of the breast in any one of them. And my own experience is that the persons whom I have had to do with with cancer of the breast have been, on the whole, people of respectable character, and not in the remotest degree likely to be affected specially in any way with syphilis. Therefore, I am sure syphilis has nothing to do with carcinoma. Of course, syphilis has no little part in certain forms of epithelioma in the mouth and tongue, or, indeed, in any mucous place in the body, because if you will only irritate a mucous membrane sufficiently long in the healthiest of men you will produce an epithelioma. If you will take a burn and irritate the scar of it long enough you will make an epithelioma, but it is only in virtue of this constant source of irritation.

Now let us look at another point. What about bad water, bad housing, and bad sanitation? Is there anything in these? There cannot be. All our great cities are furnished now with first-rate water. The insides of them have been torn out and replaced with better houses. In the old days if a man bought a house and it was suggested to

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him that he should look after the drains, he was indignant. The first thing he does now is to look after the drains. Nothing has advanced so much in our lifetime as sanitation. Insanitary conditions, then, can have nothing to do with cancer. Then we have electricity, steam, telephones, telegrams: these all come into our daily lives. Is there any chance of them acting upon cancer? None at all. It is not common sense to suppose it. They affect our nerves, and they will transform the next generation into neurotics, but they will not make them more cancerous.

What is the condition, above all others, which we find prevalent in England, and all over Europe, and other civilised places? It is the fact that during the last generation the whole mass of people have had far more to eat than they used to have in former days, and food of a far better quality. You have only to consider the enormous amount of corn that has come into this country since the corn laws. People have plenty of bread to eat, formerly they had very little. Look at the shiploads of food which come into my city of Liverpool. The shiploads of cattle which arrive into this country every day are prodigious. People now eat meat every day of their lives, whereas formerly, forty or fifty years ago, they ate meat only once a week, even if they did so then. Now the workman eats his meat every day, and a man in better circumstances eats it three times a day; he has it for breakfast, for lunch, and for dinner. Therefore I have a strong view that the increase in the amount of rich food, and notably of meat, among civilised people has something to do with cancer. Go to the out-patient room of hospitals and see the people who are cancerous. Are they poor, miserable, attenuated spectres,—tea-shrivelled people? Not at all. The generality of cancerous patients are well fed, with plenty of fat and muscle on them, and they are nearly always of a good colour until they come to the last stages. In short, as Mr. Moore, the man who introduced the operations of real efficacy into this or any other country, once said, "cancer is eminently a disease of persons whose previous life has been healthy, and whose nutritive vigour gives them otherwise a prospect of long life." On the whole, that is my experience of the cancerous person. And I will say, further, that the better the state of nutrition that person is in, the more rapid is the advance of his cancer. Well, whatever may be the other points, my own notion is that the increase in the taking of heavy food and abundance of it has something to do with this disease. I noticed a statistical point which Dr. Tatham brought out at the last meeting here, and that was the question about the male, and how cancer was increasing in him. Now, if you notice, it is the male who eats more, and who eats more richly; more food and better food is being put before him. The male's diet is increasing in point of amount and richness. I do not think women's diet has varied so much, but young men at the universities up and down the country conceive it to be their duty to eat enormously of meat. They think it has something to do with their athletic functions. Women do not. With them I do not think diet has varied very much, but the male has eaten more and more meat, and that goes concurrently with the statement that cancer is increasing with them. There has been recently put forward a theory as to the consumption of salt by Dr. Braithwaite. I read his paper with some care this morning. He drew his deductions primarily from the fact that Jews have very little cancer, and that they certainly do not eat salt pork. In the *British Medical Journal* (March 15th) probably many of you saw a very well-written article from a correspondent, in which he showed that Dr. Braithwaite had obviously not collected his statistics with sufficient care, inasmuch as this gentleman, who is evidently very intimately acquainted with the habits of the Jewish people, distinctly said it was a very great question whether they did not suffer from cancer just as much as Christians. Furthermore, Dr. Braithwaite has, perhaps, a rather happy way of getting out of the difficulty. He says, after all, if they eat much meat they must eat much salt with it. I do not think the salt theory will hold.

Let us go now to the great question of the cancer parasite, as it is called. In Liverpool I have two friends, junior members of my profession—Dr. Buchanan and Mr. Montserrat—who have worked in a quiet diligent way, and I have with pleasure watched their work and seen it. Is there a cancer parasite? Opinions vary, but I believe you have heard here that the cancer parasite is a degeneration. My own opinion, formed from the specimens of these two gentlemen, is that it is not a degeneration. I think there is a special body in cancer

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very easily recognisable when you have learnt to see it, which at all events accompanies cancer. We had a very interesting debate the other night at our Pathological Society of Liverpool, and to it there came Mr. Plimmer, from London, whose knowledge on this subject is greater than that of any man living. He defended the parasite. And there came Mr. Stiles, from Edinburgh, to denounce the parasite. I listened to them both with great attention, but I did not think Mr. Stiles had proved his case with regard to the degeneration. One thing struck me as a strong argument against it, namely, that the growing parts of carcinoma are the circumferential parts and not the centre. Now the latter is the point where the fibrous degeneration is going on. The active part of the growing tumour is at its circumference, and only there do you find these cancerous parasites. That appears to me to be entirely opposed to the very essential notion of degeneration. Another very remarkable point which should be noticed is that in the staining of these cancer parasites the stains which attach to them are those that do not attach themselves to the nuclei of epithelial cells. These two are very strong arguments to combat. But that is a question which is still under consideration. Where do these bodies come from? What are they? Do they precede the cancer? Do they follow it? None of these questions have yet been answered. Are they blastomycetes? Do they belong to the class of yeasts? or do they belong to the class rather of what we may call the animal forms of life? We are not sure of that yet. There was a strong belief that they belonged to the class of yeasts; but I think the view is now rather returning to the idea that they are rather a form of protozoa. But that is not determined. Then comes the last question: what about the infectivity? My own view is that if cancer is an infective disease its infective powers are of the very lowest type. I only saw, to my great surprise, the other day that a great body of German pathologists had given it as their general opinion that cancer was an infectious disease. I know that one gentleman, who has written a good deal about this, Mr. Roswell Park, of Chicago, brought forward a paper containing what were considered strong arguments in favour of its infectivity. I confess I could not see them. I have not seen any distinct proof of it. If it is such a very infective disease, show it to us. Well, they say, if you rub one cancerous surface steadily against another, as, for instance, the two lips of the vulva, there will be a cancer on the other side. But that is not making, *de novo*, a carcinomatous tumour; that is merely producing an epithelioma by constant irritation, which you can always do. Take any man's healthy mucous membrane or skin, and rub and worry and irritate it for a given space of time, and you will make an epithelioma; but that is not producing a carcinomatous tumour. I maintain that statements like that are not any evidence whatever. Then there are other statements about the removal of cancerous tumours being followed subsequently by cancer, the idea being that there has been infection resulting in the formation of tumours again. If that were the case, we, as surgeons, should be responsible for infecting three out of every four cases, for that is about the proportion in which recurrences occur. I saw an instance in which it was said cancer of the breast was removed, and that in the line of incision, and in the stitches, there came a cancerous infection. This is a very curious argument. If the gentleman had said the knife did it, or any other instrument used at the removal of the tumour, I could have imagined it; but where does the operator take his needles and thread from? The cancerous tumour being removed, he takes a clean needle and thread, which has never been near the tumour, and he stitches up this wound. How, then, can he say he has infected all these stitches? That sort of argument seems to me to be supremely ridiculous. Cases were mentioned of surgeons having contracted cancer. I have not heard of one, nor have I met a man who could tell me of one. If it were a question of syphilis, probably there is no one in this room who does not know of some member of his profession who has suffered from syphilis. Will you tell me of a surgeon who has died of cancer from infection? I shall be glad to hear of it if there is a case. I do not believe such a thing exists. Who are the persons like to catch it if it is infectious? Surely it is we, the surgeons who work among cancer. Therefore, gentlemen, I have no belief in the infectious nature of cancer; at all events, I think it has yet to be proved.

And now I will just say this: that in all our work in relation to this matter I do most sincerely hope that a good deal more caution will be

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observed in the pronouncements which our pathologists intend to make for the future than has hitherto been done. During the interval that has elapsed since I gave my little *résumé* some years ago there was one gentleman from whose views most important results were expected. He wrote a monograph on the subject, pointing out the wonderful bodies he had discovered. But I understand, on the best authority, that these were things which came out of the cork of the bottle containing his oil. They were cork cells. And then we have our excellent Italian friends who at that time were promising wonderful things. They were making cancers out of lemon juice, and all the rest of it. What has become of them? Nothing. During recent months M. Doyen, of Paris, discovered a new germ. What has become of it? You read your 'British Medical Journal,' your 'Lancet,' and other medical journals, and you find half-a-dozen discoveries. Where are they? They have been brought before us before they were sufficiently carefully thought out. I should be very sorry if it were so, but I begin to fear lest this great question of the investigation of cancer should be what is called nowadays "exploited." There is a strong threatening of this occurring, where the object of the gentleman who has got his new cure to announce is rather to get it first in the field than for the real interest of the sufferers from cancer. Therefore, gentlemen, I think you will forgive me for saying that I do hope that we all here to-night, and the members of our profession generally, will not bring matters before each other, and notably will not bring them before the public, until they have been so matured and consolidated and so well traced out that some truthful and definite statement, which shall not be controverted and confuted within three months, shall be brought before the public.

Mr. Pearce Gould.—Mr. President and gentlemen, the subject before this meeting to-night is a wide one, and there are others to follow in this discussion, so I propose to take up but a very few minutes of your time, and to deal only with one aspect of it. One of the parts of the subject before us is the general principles of treatment of cancer. At the present time there are two great methods of treatment, so called, in which we are interested, the operative and the non-operative. The prevailing view is that the earlier and the wider the operation is the greater is the success that will follow from it. And I think that although we all of us meet with cases which give us some little check in the confidence with which we make that statement, yet I think we should all of us agree that in the main it is true. But, sir, we must not shut our eyes to the fact that amputation is not curing cancer. It may free a man from the malady from which he is suffering to excise the affected area and a large area around it, which we believe to be, or which we think is liable to be infected. But that is not curing cancer. And the question I should like to submit to this meeting is what ground there is for hoping that operation will ever accomplish all that we wish, and what ground there is for hoping that by other methods we may be able to accomplish that desirable result. Well, sir, it has been stated, I think, by Mr. Butlin in some such form as this:—that if only every cancer could be operated upon properly when it was the size of, say, a buckshot or a pea, that our results would be absolutely different. Well, I think that is probably correct. But that time will not arrive. Nothing is more striking, I think, than the perfect absence of symptoms in the earliest stage of cancerous disease. In the lip, of course, it is seen from the first; in the tongue it makes its presence very early known, but if we take the more hidden parts of the body, such as the rectum and the uterus, it is the experience, I am sure, of every one here that the disease has often advanced very far before the most careful, watchful, and intelligent patients have had any notion of it whatever. I am constantly seeing—and I am sure all of us are—women coming up with cancer of the uterus with the story that they had not the smallest idea there was anything the matter with them until a fortnight or a month before, in whom examination reveals a most extensive malignant infiltration. So that while one rejoices in every opportunity of practising surgery at the earliest practicable moment in the history of the cancer, yet one cannot but feel that with this fact before one, the time will not arrive when we shall get *all* our cancerous patients in the initial stage of their disease. As to the other point, the non-operative treatment of cancer, or what I should prefer to call the curability of cancer, is there any ground for thinking that that is true? that cancer is, or will prove to

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be curable? We see such a long and almost constant procession of sufferers from malignant disease going more or less rapidly from bad to worse, until death comes not only as a welcome, but as a very belated guest to them, that I am afraid there is a great tendency with us all to feel that cancer is an incurable disease. I am quite sure it is not.

And why I am sure it is not is this. The cure of any disease is only brought about by the agency of physiological processes. All our much-vaunted remedies only produce their beneficial results by influencing in some way or other the ordinary physiological processes of the body. We are quite unable to introduce any new force into the body, and we can only work upon and with Nature's inherent powers. And the only hope of the cure of any disease is by observation of the methods in which Nature herself deals with that disease when unaided and uninfluenced by us at all. And just for this reason it is extremely important to recognise the fact that cancer in some cases undergoes spontaneous cure—that the physiological processes of the human body are sometimes sufficient to accomplish the perfect cure of cancer. I must admit that this is very rare, but it does sometimes occur, and if it only occurs once in a million cases it is enough to give us the assurance that with perseverance of research and effort we shall be rewarded with a discovery which shall solve this great problem. I propose, therefore, to occupy five minutes more of your time in giving you some of the examples I have seen myself of the absolutely spontaneous repair that takes place in cancer. The first example I will mention will be the repair and healing of cancerous ulcers. I will begin with a very slight simple instance of the repair. I show you this drawing of a man who was recently under my care in the Middlesex Hospital, an old gentleman over eighty years of age, who had a somewhat large scirrhous ulcer in the right breast. In the centre, far away from the edge of that ulcer, he had what you see, an island of epithelium. That was formed late on in the disease, and we watched it grow to a considerable size. Here is a picture of a woman who was in the same hospital in our cancer ward a little time ago with a very extensive cancer, which had destroyed the left breast. No operation had ever been performed on that patient; what you see is all the work of the disease, large glands in the axilla, large glands above the clavicle, with the horribly distressing solid œdema, which comes on in these cases, affecting the whole arm, and you will see there not only large islands of epithelium, but all round the ulcer epitheliation had occurred, and all the time the woman was dying of the cancer, and did die of it. That picture was taken six weeks before she died. In the next bed was a woman who furnished this drawing. There the disease affected the right breast. No operation had been performed, and the whole breast had been involved in the malignant disease. She came in with a very extensive ulcerating cancer of her right breast, the ulcer being of the size of my hand, with numerous nodules about the chest and sternum, the opposite breast, both axillæ, and also cancer of one lung. She eventually died. But a month before she died this drawing was taken. When she came into the hospital there was a little blue line all round this cancerous ulcer, and we watched it spread over that malignant disease, as it does over an ordinary ulcer in the leg. A month before she died from cancer that ulcer was entirely healed over. We made a microscopical examination of the cicatrix and skin around, and we found that the epithelium of this cicatrix was exactly similar in character to the epithelium of the surrounding skin, and if we looked at the surface only we could not distinguish that scar from an ordinary scar. But underneath that normal epidermis there was a thin layer of scirrhous growth. We had, in fact, a skinning over of a cancerous ulcer with squamous epithelium, and that is, of course, a very marked evidence of repair. The disappearance of malignant nodules is very familiar to all of us, I mean spontaneously, quite apart from any treatment. I could mention two cases in which quite extensive malignant growths have spontaneously disappeared. One of them I mentioned at the Clinical Society of London and published there; but it is worth mentioning again. It was the case of a woman who had had the right breast removed for malignant disease, and was subsequently admitted to our cancer ward with extensive recurrences and secondary deposits of cancer. When I saw her she had an extensive local recurrence, growths in the same axilla, and on the same side of the neck, a mass in the left breast, and enlarged glands in the left axilla and supra-clavicular triangle; there

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were also signs of what was believed to be extensive malignant disease in the right lung. The neck of her right femur underwent spontaneous fracture, and a considerable tumour developed in the neck of the femur. I do not think it was possible to doubt that the bone was the seat of malignant growth. While she was so ill, that we were daily expecting, and indeed hoping, for her death, without any special treatment at all, this patient began to mend and these deposits to disappear. The neck of her femur consolidated, it is true, with considerable deformity, the tumours all vanished, and she has remained well for several years now. There has been no return of any one of those deposits. I think one can fairly cite this as a case in which the ordinary physiological forces of the body dealt satisfactorily with very extensive malignant disease. The second case of a similar kind which I will mention is that of a man who came to the hospital two years ago with carcinoma of the left superior maxilla. I excised it, and he went back home. He was sent to me by his doctor some few months afterwards, and to my regret I found there was a very considerable local recurrence. The chasm which I had left in the man's face was more than filled up by a considerable fungating growth in his mouth. I sent him back to Wales, and wrote a letter to his doctor saying nothing further could be done. A few months afterwards he wrote to say the man was better, and that he would like me to see him again. He came to me seven months afterwards, and I found him then absolutely well. There was not the smallest sign or trace of any malignant disease about him. That again, I think, is a very striking instance of the power which nature has of dealing with this disease. I might take up time, sir, in quoting other cases, but it is not necessary. I may add that all the cases I have mentioned were examined with the microscope, and there is no doubt that they were cases of true carcinoma. Only yesterday, to mention one other case before I sit down, I saw a patient who was operated upon for cancer of the left breast at the Chelsea Hospital for Women. That is nearly three years ago now. She returned to that hospital several months ago (that is to say, two and a half years after the operation) with recurrences of the disease; there were two nodules over the front of the chest, two lumps in the axilla, and a considerable mass above the clavicle. The liver was enlarged to the level of the umbilicus. There was a little fluid in the peritoneum, and the surface of the liver was hard and uneven. The patient's general condition was bad; she was very ill. The surgeon under whose care she had previously been very wisely said it was a case in which no further operation should be performed, and he recommended her for admission to the Middlesex Hospital cancer ward. There she happened to come under my care. We applied no special treatment whatever, but she steadily improved, and after a time got so well that she insisted upon going out of the hospital. I sent for her to see me yesterday. She has now one tiny nodule close to the scar in front about the size of a split pea; nothing whatever in the axilla. A little gland can be felt above the clavicle, and the liver has shrunk very considerably, so that it now reaches only two inches below the eighth costal cartilage, and it can only be felt below a small portion of the right costal margin, and in the true epigastrium not at all. There is still fluid in the peritoneal cavity. Her general condition has improved quite as much as the local condition, and she is, "in herself," very much better. That is a case in which the disease has not wholly disappeared, but there has been such considerable improvement that its entire removal is certainly very probable.

Gentlemen, of course this experience is not peculiar or special at all. What happens in one hospital unquestionably happens elsewhere. Cases of cancer do get better sometimes. There are only a few such cases, but I think it is extremely important that we should all of us keep these cases very clearly and constantly in our minds, so that we may never lose heart or hope in our efforts and thoughts for the treatment of this disease, but be confident that by persistent persevering effort we shall learn the secret and be able to deal with the disease by other than surgical measures. In the meantime let us perfect our surgical measures all we can; let us employ them as early and as thoroughly as we can. I would not be understood as saying one word to call in question the value, so far as it goes, of early, wide, efficient operation for cancer; but we must not keep our thoughts fixed upon that side of the treatment and think that is to be the last word in the treatment of cancer. There will be a much better word than that spoken some day. Soon may that day arrive!

(*To be continued.*)

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THE DIAGNOSIS OF MALARIA FROM THE STANDPOINT OF THE PRACTITIONER IN ENGLAND.

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Fothergill Medallist, 1902.

Delivered before the Medical Society of London, May 12th, 1902.

SLIPSHOD diagnosis is not to be commended. We must confess, however, that, so far as the interests of patients are concerned, in the vast majority of diseases, provided the physician abstain from a too active interference with natural processes, it matters little whether diagnosis be exact or even correct. In too many diseases we possess little or no power for good. Fortunately for the reputation of our art there are a few diseases over which we can exercise absolute or relative control; and just because we possess in the case of these remediable diseases this power, it is of the utmost importance that we recognise them, that we diagnose them accurately. The disease about the diagnosis of which I propose to offer a few remarks this evening —malaria—is one of these. For it, in quinine, we possess an almost infallible remedy. Hence, if we fail to recognise malaria when it is present, and in consequence of such failure in diagnosis omit to administer the appropriate remedy, the patient suffers, and we grossly fail in duty. Contrariwise, if in some given case we erroneously diagnose malaria and treat the patient accordingly, we certainly treat the case wrongly, perhaps miss the chance of cure by more appropriate means, waste a valuable drug, and certainly run the risk of damaging our own reputation. The ability to diagnose malaria is, therefore, a matter of high practical importance.

In those countries in which malaria is endemic and common, it not infrequently happens that the infection is overlooked. As a rule, however, it is the other way about; malaria is far too frequently

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assumed to be present where it has nothing whatever to do with the case. This remark is equally, if not more, applicable to certain cases of disease which come to England from abroad. Such is the exaggerated idea of the insalubrity of tropical countries, of the universal prevalence of malaria in them, that a very large proportion of the febrile, and even non-febrile diseases coming from them to this country are diagnosed and treated as malarial. It happens far too often, both in England and in the tropics, that the practitioner, when in difficulties about a tropical patient, assumes the case to be malarial. The patients, likewise, are very prone to be content with and to endorse such a diagnosis, for they too, from one cause or another, have come to believe that in the matter of disease "tropics" spells "malaria." Somehow to many there seems to be a sort of fascination and appropriateness about the word "malaria," as if it carried with it mystery, romance, and explanation such as is said to belong to the blessed word "Mesopotamia." In the course of twelve years' experience of London practice I have seen some two or three cases only in which malarial infection had been overlooked; in the same time I have seen a very large number of cases in which malaria had been diagnosed, but in which malaria was neither present nor had anything whatever to do with the symptoms.

The latter type of case I meet with so frequently that I thought, when asked to read a paper before this Society, I could not do better than choose for my subject this very practical matter of the diagnosis of malaria as we find it when imported into England.

Whatever may have been the difficulties in the way of reliable diagnosis of the infection in the past, recent discovery has enabled us to be precise and absolutely sure about it, at all events in ninety-nine cases out of a hundred. There are few diseases so easily diagnosed; nevertheless, and although nearly every member of the profession knows, in an academic sort of way, the principles on which the points of diagnosis are founded, my experience tells me that these principles are not applied systematically, nor in actual practice assessed at their proper value. Possibly it may serve some useful purpose if, availing myself of this opportunity, I recapitulate and accentuate these principles.

The pathognomonic indications of malarial infection may be conveniently arranged under these headings:

1. The clinical.
2. The therapeutic.
3. The microscopical.

1. *The clinical diagnosis.*—There are many signs and symptoms of malarial infection—fever, anæmia, icteric tinting of skin and scleræ, enlargement of the spleen and liver, gastro-intestinal disturbance, a tendency to spontaneous recovery and to subsequent recurrence. All or any of these may lead one to suspect, but not one of them, nor the concurrence of all of them, justifies a positive diagnosis of malaria. None of them, and no grouping of them, is pathognomonic. They are present separately or variously combined in many other morbid conditions. They may justify in any given case a suspicion, but not a diagnosis, of malaria.

There is only one clinical symptom that justifies a positive diagnosis; this is periodicity, periodicity of a particular type—tertian or quartan periodicity—a rhythmical recurrence of symptoms every forty-eight or seventy-two hours. In no other morbid condition are these to be found and wherever well marked malaria is present.

In malarial disease the appearance of quotidian periodicity is common enough; but it is only the appearance. It is very doubtful if true quotidian periodicity ever occurs in malaria; if it does it is certainly very rare. The parasites of malaria are either tertian or quartan in habit, and many of the symptoms they give rise to are correspondingly tertian or quartan in their recurrences. Multiple infection by two kinds of parasites, or by two or three generations of the same species of parasite, may give rise to double tertian or treble quartan infection with diurnal recurrence of symptoms; but this pathologically, whatever it may be clinically, is not a true quotidian periodicity. It is common enough in malaria, but I would insist it is not pathognomonic of malaria, for a similar quotidian periodicity is very general, in fact is usual, in almost all febrile complaints. Therefore to make quotidian periodicity a ground for a diagnosis of malaria is wrong. More than that, it is most dangerous, and is, perhaps, the commonest cause of erroneous diagnosis of malaria. Experience has made me formulate a rule for myself which I commend to you—have absolute confidence in tertian and quartan periodicity as a basis for a

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diagnosis of malarial infection, but distrust, or rather absolutely disregard, quotidian periodicity.

In tropical practice I have seen more mistakes from relying on quotidian periodicity as a ground for diagnosing malaria than from any other cause. The temptation to attach weight to it is often very strong, but I would again urge on you to harden your hearts against it.

When a patient from the tropics comes to you with a story of evening rigor, rise of temperature to 103° or perhaps 104°, and a profuse sweat, the idea of malaria naturally presents itself. Most probably the patient tells you he has malaria. And if one finds the patient anæmic, and that his spleen is large, perhaps very large, the diagnosis of malaria seems to be clenched. But people abroad get splenic and spleno-medullary leucocythæmia, and other diseases in which the spleen is enlarged, as well as people in England, and such patients often have marked quotidian fever. These leucocythæmic and other kinds of enlarged spleen cases I frequently see on the supposition that they are malarial. Had the patients never been abroad probably the idea of malaria would never have been entertained.

Another type of case in which there is enlargement of the spleen associated with anæmia and quotidian fever I occasionally see, probably because it, too, is sometimes supposed to be malarial. In a sense the cases I refer to are malarial—or, rather, post-malarial. I allude to what is known in Assam—where the condition in certain districts is very common and very fatal—as Kalaazar. The same condition occurs in other malarious countries, however. The patient gives a history of malarial attacks, which at first were controlled to some extent by quinine. After a time the recurring attacks became more frequent and less amenable to quinine. Finally the fever resisted treatment, became quotidian, the spleen becoming permanently enlarged, the anæmia intense, the skin sallow or even melasmic, and ultimately, in spite of treatment, the patient, if not cut off by some intercurrent disease, dies of pure anæmia. This disease, although possibly induced by malaria, has long ceased to be malaria—is, in fact, a post-malarial cachexia, brought about by organic changes in the blood organs, and induced possibly by some additional and distinct infection attacking organs already damaged by antecedent malaria.

I find that the presence of enlarged spleen is a bait that few resist when attempting the diagnosis of a quotidian fever in a patient from the tropics. The combination is so suggestive. But we must bear in mind that many people in the more malarious districts in the tropics have, though free from active malarial disease, chronically enlarged spleen; and we must also bear in mind that the possession of an enlarged spleen is no protection against other diseases, and that enlarged spleen is symptomatic of many diseases, febrile and other. I have several times seen ulcerative endocarditis in patients with spleens enlarged from antecedent malaria; a coincidence merely. The same occurrence I have often seen in phthisis and in many septic conditions.

If quotidian fever associated with enlargement of the spleen is to be distrusted as a combination indicative of malaria, still more so is quotidian fever associated with enlargement of the liver. The latter combination usually means abscess of the liver. Anyone who has had much experience of tropical practice must know that this disease is often, I might say generally, at all events in the earlier stages, diagnosed malarial fever. Osler says he has seldom seen an abscess of the liver in which the patient had not been liberally dosed with quinine. This is my experience likewise. I have seen a medical man make the mistake in his own person. The gentleman I refer to had been many years in India. He fell ill, and was invalided home. He had every afternoon a smart shivering fit, a rise of temperature to 103° or over, followed by drenching perspiration. He took large doses of quinine daily for many weeks. Finally, becoming very ill, he sent for me. I found the liver enlarged, tender, the spleen not enlarged, or only slightly; these facts, with the quotidian fever and other points, satisfied me that there was an abscess in the liver. But I could not convince the patient. He asked me to make a blood examination. I did so after suspending the use of quinine for a week to give the hypothetical plasmodium a chance to get into the peripheral circulation. The result, so far as the presence of the parasite was concerned, was negative. Next, he insisted on another week of larger doses of quinine, and only then, and after much persuasion, did he accept my diagnosis and permit Mr. Godlee to open the large abscess in his liver. Fortunately he got well, but

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his conversion to the fallibility of quotidian periodicity as a diagnostic mark of malaria was somewhat dearly purchased.

Strange to say, syphilis, both secondary and tertiary, is not infrequently, on the strength of an associated quotidian fever and a residence or visit to a malarious country, diagnosed malaria. Some years ago I was consulted by a gentleman who had resided for many years in a mildly malarious country. The story he told me was that for over a year he had been suffering from ague. The attacks, he said, were quotidian and very severe. He had taken large doses of quinine and of arsenic, had visited hydropathic and similar health establishments, and done everything he could think of to get well. As soon as he told me the story of his treatment, for reasons which I will state by and by, I was quite sure he was not suffering from malaria, and proceeded to endeavour to ascertain the cause and nature of his fever. In the course of my examination I found his liver very much enlarged, studded with irregular firm masses so big and hard that I thought they were probably malignant. I asked if anyone had examined his liver before. He told me no one had done so recently; everyone had accepted his own diagnosis of his case (a very common practice, I am sorry to say), for, said he, of course he knew all about the symptoms of fever much better than any doctor, in this country at all events, could tell him. In going further into his history I ascertained that many years ago he had a chancre. In the hope, but I confess not with the expectation, that the lumps on his liver were syphilitic and his fever symptomatic of softening gummata, I put him on iodide of potassium. Two days later he called on me saying the medicine had given him headache and coryza, and so upset him that he had stopped taking it. After a good deal of trouble I persuaded him to double the dose. A week later he again called; this time with the news that his fever had vanished, his appetite had returned, and that he felt a new man. Gradually, under large doses of iodide, the irregularity and enlargement of the liver disappeared, and he got quite well.

Some time ago I saw in consultation a young gentleman who had been travelling in unhealthy countries, and who, soon after his return, had fallen sick with fever. He insisted to his medical attendant that it was a malarial fever contracted in his travels, and persisted in taking quinine. Not improving, and still believing that his fever was malarial, I was asked to see him. Beyond a quotidian fever there was no evidence of malaria about him; but we elicited a history of a sore on the penis some months before. Having been told by the doctor he saw then that the sore was a soft sore and of no importance, he had not thought it worth while mentioning this circumstance before. We changed the quinine for mercury, and in a very short time the fever vanished.

Septic absorption of pent-up pus is a well recognised cause of quotidian fever not unfrequently attended with marked rigor and diaphoresis. Sometimes the most careful examination fails to determine the situation of the pus. Now, if suppuration of this description occurs in an individual who has resided in malarious countries the case is almost sure to be diagnosed malaria. I have seen many such cases. Some time ago I was asked to see a young gentleman who for a long series of weeks had been the subject of a quotidian ague-like fever. He had travelled abroad. He had been seen by a number of eminent medical men, each of whom had given a different opinion, and prescribed an equal variety of treatment. I was asked to see the patient with the idea that the case might be malarial. I had no difficulty in pronouncing definitely against such a diagnosis, but what the nature of the case might be I did not pretend to know. Months later I met in the street a medical friend who devotes himself to throat and nose diseases. In the course of conversation he said, "By the way, you saw some time ago a Mr. So-and-so with Dr. ——?" "Yes," I said, "what about him? How is he? Is he dead yet?" "Far from that," my friend replied, "he is quite well. Some time after you saw him I was asked to examine his nose, which was bothering him, and by way of cleaning the passages preliminary to examination I syringed his nostrils with a warm alkaline solution. I did nothing more, and could see nothing wrong. But since that time he has had no recurrence of his ague-like attacks, and he is now quite well."

Some years ago I was asked to see a retired member of the Indian medical service, said to be suffering from malarial fever. I was told he had been treating himself, but had become alarmingly ill. On arriving at the house I found the patient

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insensible, moribund in fact. Another medical man had been called in by the wife, but neither he nor I for a time could get any light on the case. The wife could only tell us he had suffered much from fever both in India and since his return to England. We could find no evidence of enlargement of the spleen or other indication of visceral disease, such as might explain the situation. In looking round the room for bottles or other evidence of poisoning we came across a very ancient gum-elastic catheter. This gave the hint. On examining the patient's perinæum we discovered a huge abscess. This was freely incised; too late unfortunately to save the patient.

Pyelitis, catheter fever, pylephlebitis, empyema, phthisis, middle ear disease, elephantoid fever, the fever of early leprosy, the fever sometimes associated with gall-stones, these and many similar conditions occurring in patients from abroad, because they are accompanied by an ague-like quotidian fever, and because they occur in individuals who have been abroad, are constantly being diagnosed malaria and treated as such. Therefore I say, in approaching the diagnosis of a pyrexia in a possible malarial, while regarding tertian and quartan periodicity as absolutely conclusive, attach no weight whatever to quotidian periodicity.

2. *The therapeutic diagnosis.*—Whatever may be the case in certain malignant infections in tropical countries, quinine, properly given, so far as my experience goes, never fails to arrest a malarial attack occurring in this country, and this within forty-eight, or, at most, ninety-six, hours of its administration. Therefore, a periodic or remitting fever that persists after adequate dosing with quinine, say for four days, is not malarial.

Every one believes, or says he believes, in the anti-malarial virtues of quinine, but it is strange how little confidence some practitioners have in its diagnostic value. Thus I frequently see in consultation cases of fever presumed to be malarial which have been taking quinine in large doses for weeks, or even months, and yet no suspicion about the correctness of diagnosis had been entertained. When asked to see a case of supposed malarial fever of a persistent nature my first question usually is, "Has the patient taken quinine, for how long, and in what doses?" If I am told, as is often the case, that he has had large doses for many days, I feel sure, without seeing the case, that it is not one of malaria. In this country, if any of you are called on to treat a fever you suppose to be malarial, and if this fever resist quinine properly given and in full doses for three or four days, my advice to you is to revise your diagnosis; the fever is not malarial. I could quote many cases in illustration. It is hardly necessary to do so. The case of liver abscess I have already related is a case in point.

There are two important matters, however, which require to be attended to in applying the quinine test. First, the dose must be adequate. Second, the quinine must be absorbed.

In many instances quite an insignificant dose will check an ague. Many years ago, while in practice in the island of Formosa, I ran short of quinine and had to economise my very limited stock. With this object I experimented to ascertain the minimum dose that would check an ague, and ultimately came down to half a grain three times a day. My Chinese patients in several instances said they were cured by this insignificant amount—so potent is quinine in some cases of malaria. This winter I had in hospital a patient suffering from double benign tertian infection. By a properly timed small dose of quinine it is sometimes possible to eliminate in such a case one group of the parasites without injuring the other group, and so to convert a quotidian ague into a tertian. I wished to demonstrate this to the students at the School of Tropical Medicine. I gave the patient three grains of quinine an hour or thereabouts before an expected paroxysm. Small though the dose was it killed out not only the group of parasites I proposed to eliminate, but the other group as well, and contrary to my intention cured for the time being the patient.

Quinine as against the malaria parasite is a very potent drug, but for reliable diagnostic, as well as for theurapeutic, purposes it must be administered in much larger doses than those I have just mentioned. I would say that if a fever in this country resists ten grains of quinine, given three times a day for four days, it is not malarial.

The dose of quinine should not only be adequate, but we must assure ourselves that it is absorbed. I am quite certain that many of the failures to control malaria by quinine are attribut-

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able to neglect of this essential. A catarrhal state of the gastric mucous membrane is usual in severe malarial attacks; such a condition is very unfavourable to absorption. Persistent vomiting is also common enough; food and drugs are alike rejected. To get the therapeutic action and diagnostic value of quinine in such circumstances, the drug must be given hypodermically, or by enema.

Quinine nowadays is often given in tablet, or coated pill form; a most dangerous method in serious cases, and a most unsatisfactory one for diagnostic purposes. For some time quinine tablets have not possessed the flint-like hardness that used to characterise them a few years ago; they are now more soluble, but even lately twice I have seen them fail to be absorbed. In one case the patient developed a severe malarial attack on his way home from the Gold Coast. He took quinine tablets, and persisted in their use after his arrival in London. So much quinine did he take in this way without benefit, that both the patient and his medical attendant lost confidence in the drug, and the patient came very near dying. Three or four intramuscular injections of the acid hydrochloride broke the fever, and the patient quickly recovered.

In giving quinine, therefore, whether for therapeutic or for diagnostic purposes, we should assure ourselves that it is absorbed.

3. *Microscopical diagnosis.*—Of the three means for diagnosing malaria this is the most satisfactory, provided it is undertaken by one with large experience. A negative result by the amateur is valueless; a positive result is not much more to be trusted. It takes a large amount of practice to be able to recognise all forms of the malaria parasite, or to discriminate between them and vacuoles in the red corpuscles in fresh films, or dirt in dried and stained films. Articles in the medical papers by amateurs in malarial blood examination sometimes supply amusing reading. The frequency with which such writers encounter malarial rosettes and flagellated bodies, and free flagella, forms of the parasite least frequently met with, is very suggestive. Free pigment—in reality dirt—they often see. White blood-corpuscles I have known described as malarial parasites; blood platelets, as spores or sporulating parasites; cotton fibres or fibrin, as flagella. No one without some knowledge of the histology of normal blood, and much experience of microscopical examination of malarial blood, should attempt to write on the subject of the malarial parasite, much less attempt responsible diagnosis of malaria with the microscope. The thing is easy enough when one has been properly taught, and after large experience. Then the microscope is invaluable in diagnosis; but those who have not been so taught, and who have not had much experience, are eminently unreliable —they are often worse than useless. Therefore, for the majority of medical men, if they wish to have blood examined for the purpose of diagnosis in regard to the presence of a possible malarial infection, the services of an expert are necessary. But although he may not be able himself to diagnose malaria with the microscope, every medical man should know how to prepare blood films suitable for such an examination. Very singular notions are entertained about this. I have sometimes had blood sent me in capillary tubes, such as are used to hold vaccine. I have also had sent me blood so thickly smeared on a slide that the preparation would not transmit light. I have also had sent me, from a distance, an ordinary slip and cover glass wet preparation, with a request to examine it for malaria. Blood transmitted in any of these ways is useless. Unless the fresh blood can be examined at the bedside, or within an hour or thereabouts of preparation, dried films should be made and subsequently transmitted to the laboratory for fixing, staining, and microscopical examination.

There are many ways of preparing malarial blood films. I have tried most of them. The best, I believe, is that described by Drs. Christophers and Stephens, or some modification of this. An ordinary microscope slip is carefully cleaned with alcohol. The patient's finger tip or the lobe of his ear is similarly cleansed. Finger or ear is then pricked with a clean and rather long ordinary sewing needle. A drop of blood is pressed out and, as it may contain epithelial *débris*, wiped away. A second but very minute drop, the size of a large pin-head, is then expressed, and touched lightly (the skin must not be touched) with the clean surface of the slip about an inch from one end. Immediately the shaft of the needle is laid transversely on the droplet of blood so transferred to the slip. After waiting a second or two for the blood to run out by capillarity between the surface

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of the slip and the needle, the latter, held between finger and thumb, is drawn along the surface of the glass. In this way, with a very little practice, a beautifully fine and even film is produced. Half a dozen such films should be prepared. The single prick suffices as the films can be made very rapidly one after another. After they have dried they should either be wrapped in very clean tissue-paper or placed in a dust-free slide-box, and transmitted to the laboratory. Here they are fixed with alcohol (five or ten minutes) and subsequently stained and examined.

For purposes of diagnosis I use either borax, methylene blue, or dilute carbol thionine. These are very reliable stains, keep well, act rapidly, and give sufficiently good results. Undoubtedly one of the several modifications of Romanowsky's stain, when successful, gives a more brilliant picture; but they are somewhat unreliable, and unless the particular sample of stain employed is in frequent use, and known to be good, this method is not advisable for ordinary purposes of diagnosis. Leishman's modification of the Romanowsky method gives very beautiful results, and has the advantage of not requiring a preliminary fixation of the blood with alcohol, but it too has the objection of unreliability in ordinary clinical conditions. Malarial blood should not be fixed by heat.

In stained films, or in fresh blood, it is practically always possible to find the parasite, at all events in those cases of malarial infection that come to England. But this only on one condition, and this condition I would impress on you—the patient must not have taken quinine recently. You may find certain forms of the parasite, the crescent, for example, after full dosing with quinine, but the usual fever-causing forms, as a rule, rapidly disappear after administration of the drug. Therefore, if you wish to have a reliable microscopic diagnosis in supposed malarial fevers, do not give quinine, or suspend its use for some time, before preparing the blood slides.

It is difficult to over-state the value of the microscope in the diagnosis of malaria. I could quote many cases in illustration. I shall mention two only; one showing what may be gained by employing it, one showing what may be lost by not employing it.

Some months ago I was asked to see a patient in the Great Northern Hotel. I was told he had just returned from abroad and was very ill. He had been sent by Dr. A. Scott of Brighton, who, suspecting malaria, advised, as the patient was passing through London, *en route* for his home in the country, that I should be consulted. I found an emaciated, delicate-looking lad with an appearance suggestive of phthisis. I tried to get from him his history and symptoms, but he would not converse. On being asked a question he would smile in a vacant sort of way, and perhaps, after a long pause, emit a monosyllabic answer, but it was impossible to extract any useful information from him. Manifestly he was off his head. His parents informed me that he was twenty years of age, and had been in a cattle and fruit ranche in Shaster County, California, since the spring of 1900. He got on well on the ranche, liked the life, and kept his health till July, 1901, when, judging from his letters home, he appears to have had a malarial attack. For some time after this the parents received no news of their son. Becoming anxious, they telegraphed on October 9th for information, and received for reply: "Son has changed for the worse, but doctor gives hope." Immediately on receipt of this alarming telegram they set out for California, and, on arriving at the ranche, found that their son had been sent on October 5th to the State Lunatic Asylum at Napa. Here they found him on October 30th in a deplorably dejected condition, emaciated, and so weak that they feared he would not stand the journey home. His disease was stated to be melancholia and mania. On the way home he was better and worse. Twice or three times on board ship he had feverish attacks, which came on about 10 p.m. and lasted till 2 a.m., and for which the ship's doctor gave him some small white tablets with an A stamped on them, doubtless antipyrin or antikamnia. This was the story I got on November 19th from his parents. On examination I found the patient free from fever; his lungs were healthy, but his spleen was enlarged and slightly tender. I made films of his blood, which I examined the same afternoon. They showed many malarial crescents, and an abundance of the fever forms of the malignant tertian parasite. The patient was removed to St. George's Hospital and placed under Dr. Ewart. The same evening he had some fever, but full doses of quinine rapidly cleared his blood of the fever forms of the parasite.

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It was some weeks, however, before the crescents entirely disappeared. Under quinine the mental and physical condition gradually improved. There was no more fever, and he was discharged a month later free from malarial symptoms, but still somewhat slow in answering questions, and still mentally debilitated. I have little doubt that had the microscope been used earlier the true nature of this painful case would have been ascertained and suitable treatment instituted. Though employed dangerously late, I believe the microscope saved this lad's life, and perhaps his reason.

An officer of a liner trading to the East was brought on a stretcher to the Seamen's Hospital, Albert Docks, on July 30th, 1901. The history received with the patient was that his ship had just returned from the Mediterranean, where it had been running between Brindisi and Port Said, and that he himself had been in perfect health till three days before admission. He had never suffered from malaria. About that time he was walking about, but looking ill, and was sent to his bed by the ship's surgeon. Presently he vomited blood, and passed large quantities of blood by stool, and, continuing to do so, was taken to hospital. On admission he was collapsed, with a temperature of 102·8°. Towards evening the temperature fell to 98·4°; hæmatemesis and melæna persisted. He had much abdominal pain, temperature fell to 96·4, the pulse became rapid, and he died next day. At the post-mortem examination the spleen was found slightly enlarged; the pancreas intensely congested, and containing minute extravasations of blood; the mucous membrane of the stomach and upper part of the small intestine was similarly congested, and studded over with ecchymoses. At the time the case was supposed to be one of hæmorrhagic pancreatitis. Pieces of the pancreas and intestine were preserved for microscopical examination. Owing to press of tutorial work at the School of Tropical Medicine, Dr. Daniels was unable to examine or make microscopical sections of the organs. But when, at the conclusion of the session, he had leisure to do so he was astonished to find that the capillaries of the pancreas and intestinal mucosa were packed with sporulating malignant tertian parasites. Drs. Ross and Daniels have recorded and illustrated this very interesting case in the *Journal of Tropical Medicine*, February 15th, 1902. None of us suspected malaria. The patient had not resided in any malarial place. There can be little doubt, however, that while his ship lay alongside the wharf at Brindisi, or, perhaps, during a short run on shore, this gentleman was bitten by infected anopheles, and thus received his fatal illness. Had his blood been examined earlier in the disease, or even within an hour or thereby of his admission into hospital, the true nature of the case could have been recognised, and the patient's life, perhaps, saved by timely hypodermic injections of quinine.

In rare instances and at certain times in a considerable proportion of cases, the parasite, or its product melanin, even in severe infections, may not be detectable in the peripheral blood, and, as I have mentioned, even small doses of quinine, therapeutically insufficient doses, may cause its disappearance temporarily, or, in the case of cure, for good. Even in these circumstances it would seem that the microscope may be relied on for a diagnosis.

It has been known for some years that during, and for some time after, malarial attacks the leucocytes are greatly diminished in proportion to the red blood-corpuscles. The diminution, however, is principally in the polymorphonuclears and lymphocytes, so that there is a relative increase in the large mononuclears. Drs. Christophers and Stephens, fixing their attention on this point, have marked out a method by which it is possible to give a fairly reliable diagnosis of actual or recent malarial infection, and this no matter whether quinine has been administered or not. The increase in the mononuclears, they state, is principally during the apyretic intervals, setting in as soon as the temperature begins to fall. They say, in a report to the Royal Society, that "an increase beyond 15 per cent. is proof of an actual or recent malarial infection."

Captain Leonard Rogers, I.M.S., after a very careful investigation of the point, endorses (Brit. Med. Journ., April 5th, 1902) this opinion. He concludes: "An increase in the large mononuclears to about 12 per cent. and upwards, especially during the remissions of the temperature, strongly indicates malaria."

Dr. Daniels, Superintendent of the London School of Tropical Medicine, has paid a great deal of attention to this point. In reply to a request for a statement of his views, he writes:

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"*Leucocytic variation after malaria.*—In all forms of malaria after, and to a smaller extent during, the fever, the large mononuclear leucocytes are relatively increased to the other leucocytes.

"This increase may be slight, and in certain cases has been so slight that they only formed 15 per cent. of the leucocytes. Commonly, after malaria they vary from 25 to 30 per cent., and occasionally are over 40 per cent.

"The increase does not seem to be dependent on the species of parasite, on the number of parasites present during the acute attack, or on the severity of the symptoms or sequelæ. It is maintained for a period varying in different cases from two or three weeks to at least as many months, and slowly diminishes.

"It is not affected by quinine, either in large or frequently repeated doses.

"With some exceptions it affords a satisfactory proof of the probable recent presence of parasites.

"In normal blood it is rare to find more than 12 per cent., though I have seen as high a percentage as 16. In typhoid and Malta fever I have seen a similar increase, but it is exceptional. It is said to occur in smallpox. In certain diseases, particularly scurvy, there is also a marked increase; but in this disease there is also leucocytosis, and a more marked increase in the lymphocytes.

"The absence of this variation is of more value as a diagnostic point than its presence. As far as I have worked with the method, the only exceptions I have seen were when an intercurrent disease, such as pneumonia, which is associated with increase in the polymorphonuclear elements, also occurs. In other cases it has shown itself to be a reliable method for the exclusion of recent malaria.

"Working for three sessions with classes of students of twenty odd, many of whom, coming from the tropics, have suffered from malaria, this test has readily discriminated those of the students who have recently had malaria from those who have not.

"It has proved of value in differentiating cases of yellow fever from malaria (Low and Gray in St. Lucia).

"It has proved of value in differentiating the hyperpyrexial fever of West Africa from malaria (Sims, Congo).

"Though not of the same value as the presence of parasites, or of pigmented leucocytes, or recent pigment in the organs, it is of much higher value as a test of recent malaria than splenic tenderness or enlargement, and is of particular importance in cases where, either in the natural course of events, or after the administration of quinine, there are no characteristic symptoms.

"It is probably of about the same value as the agglutination test in typhoid with moderate dilutions of the serum, say 1 in 20.

"The main objection to the method is the difficulty that is sometimes present in deciding which are large mononuclears and which are lymphocytes. After actual practice this difficulty is small, and a column for undecided or doubtful leucocytes will be found useful at first. After a short time this will be, in the majority of cases, little used. In these determinations, however, there is a certain personal equation, so that counts made by different individuals vary within small limits."

These are Dr. Daniels' opinions, and, being based on large experience, I attach much value to them.

A few weeks ago I saw in consultation a gentleman recently returned from abroad. On the previous Saturday he was suddenly seized with a rigor and fever; on the Sunday he had no fever; on the Monday he had another paroxysm, and his temperature kept a degree or two above normal during Tuesday. I saw him on Wednesday morning. His temperature was then normal. He had received a few small doses of quinine. The tertian periodicity of the fever and a slightly enlarged spleen indicated a malarial attack, but to make certain I examined a film of his blood. As might have been expected from the quinine he had taken, there were no parasites, but a count of the leucocytes gave 20 per cent. large mononuclears, thereby confirming the clinical diagnosis. It is under such circumstances that a study of the leucocytic counts and variation will be of service in helping to correct diagnosis, and also in discriminating malaria from such conditions as abscess of the liver, pneumonia, and so forth, in which there is a leucocytosis, from typhoid, in which the lymphocytes are increased, and from other diseases with similar characteristic blood changes.

It is difficult to over-rate the practical value of blood examinations; only last week I had a striking illustration of this. A lady from India consulted

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me about what she described as an enlarged spleen. She had lived in a malarious district, and in years gone by had suffered a great deal from malarial fever; less so of late years. Medical men in India had told her she was "saturated with malaria." She came home on account of her health, especially on account of the enlarged spleen. She was rather emaciated, anæmic, and sallow. On examining the abdomen I found the pelvis and right side of the abdomen filled with a large, somewhat movable rounded tumour. There was a notch low down on the right margin of the tumour, but there was an equally pronounced notch, if not two notches, on the left border. The tumour was not connected with the liver, and the whole of the *left* hypochondrium was resonant and soft. No indication of a spleen could be detected in the usual situation of that organ. The patient was positive, however, that the tumour began in the left hypochondrium, and that it was only recently that this part had become soft. Feeling puzzled about the case, I sent the patient to Mr. Meredith for examination of the pelvic organs, and, if thought desirable, for exploration or operation. Mr. Meredith was equally puzzled. The uterus and ovaries were not involved. We advised the patient to go to a nursing home with a view to exploration. At our first interview I had made some films of her finger blood, but being busy did not overtake their examination till two days later. You may judge of my astonishment, and of my thankfulness that our operative suggestions had not gone further, when, instead of finding a slight increase of large mononuclear white blood-corpuscles, indicating recent malarial infection, I found that our patient was suffering from advanced splenomedullary leucocythæmia. The tumour we were puzzled about was undoubtedly an enlarged leucocythæmic spleen, which had in virtue of its weight and size become displaced.

We sometimes get assistance in concluding for or against malaria from considerations of time and locality. It is very commonly believed that a malarial infection may last a lifetime. Thus the old Indian, many years after he has come home for good, may tell us that he has recurrences now and again of a jungle (that is malarial) fever contracted in youth. I do not believe in the malarial nature of such attacks. I have never been able to find the parasite in any such case. My belief is that malarial infection does not persist after three years from the date of invasion. Of course a patient can become re-infected; that is another affair; but I do not think the duration of any given infection exceeds three years. I will not be positive about this; we want more observations. But in my experience a malignant tertian tends to die out in from eighteen months to two years, a benign tertian in about two to two and a half years, a quartan in about three years. We know that other protozoal organisms having asexual and sexual phases require resuscitation by passing through the sexual phase every now and again; otherwise they become extinct. I believe the same to be the case with the malarial parasite. Other people besides the old Indian get feverish colds and different kinds of short febrile seizure, but because they have never been in India or Africa they call them influenza or what not. Had the old Indian never been to India he would do the same. He is rather proud of his jungle fever, and sticks to it longer than it sticks to him. While alive to the possibility of my being wrong in my belief, I am inclined to regard any fever which occurs three or more years after the patient has been away from the possibility of fresh malarial infection as non-malarial.

If a patient has never been to a malarial country he cannot get malaria unless by artificial or abnormal means. In the malarial belt there are many non-malarial places—Malta, for example. A chronic or recurring fever from Malta in a person who has not visited a malarial place is probably Malta fever or enteric, certainly not malaria. Similarly, Barbados is not malarial. Therefore a chronic or recurring fever in a patient from Barbados who has never visited a malarial place cannot be malarial; most probably it is the fever symptomatic of some of the lesions in filariasis so common in Barbados. Commonly enough, because such fevers are ushered in by severe rigor, these filarial fevers are locally known as "ague." This is a misnomer, they have nothing to do with malaria.

I have seen a good many reputedly malarial patients from the Fen districts and other formerly malarious spots in England. On critical examination not one of them has turned out to be malarial. They were assumed to be malarial because there was some periodicity about the fever, perhaps enlarged spleen, and because they came from localities which were formerly malarious. I have

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never seen a case of indigenous English malaria. I believe the plasmodium has died out in England, like the wolf and the wild boar.

In concluding these rambling and disconnected, but I trust practical, remarks, I would again impress on you the importance of a thorough reliance on the three classical methods of diagnosing malaria: the clinical—tertian and quartan periodicity; the therapeutic—the curative effect of quinine; and the microscopical—the detection of the parasite or its products in the blood and the characteristic leucocytic variation. A thorough and intelligent application of one or all of these methods seldom, if ever, fails to lead to a correct diagnosis for or against malaria. At the same time I would warn you that although a positive diagnosis of malaria may be indicated in some given case, the presence of malaria does not exclude other diseases. On the contrary, malaria often predisposes to other infections. Similarly, the explosion of a latent malaria is favoured by other infections, especially typhoid and tuberculosis.

CANCER: ITS ORIGIN, NATURE, AND GENERAL PRINCIPLES OF TREATMENT.

(*Debate continued from page* 96.)

MR. MARMADUKE SHEILD.—Mr. President and gentlemen, I was not particularly anxious to take part in this debate to-night, because I have long felt that in the medical profession, with regard to the origin of cancer, there has been a great deal too much talk and discussion, and too little real knowledge. I understood that this debate was to deal, if possible, with the origin of cancer; and when I was asked to come here I felt myself so ignorant of the matter that I wished to avoid speaking rather than to appear before you. But be that as it may, I am here to-night, and I wish as far as possible to indicate in what directions I believe our investigations should proceed in order, if possible, to elucidate the phenomena of this mysterious malady. It has long been my belief that the way in which our study should proceed in this matter should be from very simple beginnings to the greater ones. I suppose there is scarcely one of us in this room who, if he critically examines his skin, will not find upon the surface of his body some little warts or fibromata, and perhaps fatty tumours. We know nothing more of the origin of these than we do of the origin of cancer. No one can tell, for instance, why on one man's body a number of fatty tumours should occur. You have heard of such cases where a man in perfect health will be covered with dozens of lipomata; again, no one can tell certainly why a child should have nævoid tumours upon him, and another boy cancellous exostosis. These are all as mysterious as cancer, and quite as worthy of our study. I have often thought we may go further back than this, and see how very important a careful investigation and prolonged study of the diseases of plants and trees and vegetables may be. What a suggestive matter is the formation of cellular tissue around the parasite of the oak gall, those extraordinary excrescences which form about a parasite in the oak leaf, and those tumours on the trunks of beech trees, which are rudimentary buds growing in the bark. Then there are the enormous excrescences around birch trees, which are supposed to be due to embryonic buds. I think a prolonged study of these subjects is of the first importance to scientists. How little has anybody studied the morbid growths of the lower animals, I mean in such lowly beings as molluscs and crustaceans. I believe molluscs, and especially fish, are very free from all forms of tumour growth, but this is a matter which has not been investigated with the thoroughness which I believe scientific explanations demand. All these considerations are of the highest importance, and I believe that results of an important nature might spring from prolonged investigations of this kind. I feel confident that with all this, we need a most careful correlation between pathological and clinical work. The subject is of such enormous importance to mankind, that I think the time has now arrived when a very thorough investigation should be made into all subjects connected with tumour growth, and that the most careful and complete correlation should exist between clinical observation and pathological research. But the remarks I am now making are to me full of difficulty, because I feel the futility—to use no stronger term—of standing up here and advancing theories as to the laws which govern cellular

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development. To say there may be fluids which permeate the body and regulate proliferation of cells, or to describe organisms which at present are quite undetermined, one could stand here by the hour and do that, but what good result would be obtained? The point which I will next pass on to is one which I am sure anybody who is interested in surgery in this room will greatly appreciate, and that is the necessity of a more accurate and profound classification of the varieties of diseases which now pass under the name of cancer. For instance, is a slowly growing epitheliomatous cancer in the face of an old man the same disease as a rapidly proliferating carcinoma of the breast of a woman? Then there is that pathological puzzle melanosis which, I think, is one of the greatest mysteries we experience. A mole, a melanotic spot, enlarges without obvious cause, and the body becomes permeated with pathological pigmented growths. Are we right in assuming that that is the same disease as cancer of the rectum, or of the face, or of the stomach? With regard to cancerous growths of the skin, they are so diverse in their microscopical characters that I have repeatedly been puzzled how to classify them. Some of you will recollect that several years ago I published a case, with illustrations, in the 'Lancet,' of a young man who, from exposure to the sun's rays, got scores of epitheliomatous ulcers over his face and neck; he was one mass of freckles and ulcerated growths. These ulcers were excised in a series of operations, and two years afterwards he was quite well. I have called them epitheliomatous ulcers. They were examined by Dr. Galloway with scrupulous care, and he pronounced them epitheliomata. I never saw anything exactly like them. Turning to sarcomata of the skin, the diseases passing under that name are so various that I am sure at least three or four different conditions are described by dermatologists under one name.

May I speak now of the remarkable instances of spontaneous disappearance of growths reputed to be certainly cancerous? Mr. Pearce Gould has, perhaps, already taken the subject out of my mouth by mentioning some extraordinary cases of spontaneous cure and healing of cancer of the breast. But I hope I shall not be wearying you if I draw your attention to some remarkable cases of tumours within the abdomen which have disappeared under my own observation. The first case which I will mention is as follows:—Some years ago, under the care of the late Dr. Cavafy, there was a young woman who had dysphagia and ascitic fluid in the abdomen, a remarkable combination in a young woman. The bougie which was passed was arrested close to the orifice of the stomach. The abdomen was opened, and surrounding the œsophageal end of the stomach was a firm pink nodulated tumour which infiltrated also the fissure of the liver. The stomach could not be drawn to the abdominal wall to do gastrostomy. The case seemed hopeless, and accordingly the abdomen was closed after the evacuation of the fluid. She was greatly benefited for a time, but she gradually sank, and died some months after the operation. I saw the post-mortem examination, and the growth had somewhat increased, and we said it was a remarkable case of cancer. We pronounced it to be cancer. Portions of the growth were taken and examined, and it was found not to be cancer at all. There was no trace of cancerous disease. It was only a lowly organised form of fibrous tissue similar to that of a scar. You may say at once there was ulcer of the stomach or duodenum. There was nothing of the kind; we looked carefully for it, and the interior of the stomach and the whole of the intestine was healthy. Again, I have now living two cases where gastro-jejunostomy was performed. One of these is well three years afterwards, and the other seven years afterwards. In the one there was as typical a cancerous pylorus, both to sight and touch, as you could imagine, but they are both now fat and well and ruddy, and no trace of tumour can be felt. There was another case at St. George's Hospital last year, and some who are here may remember it. It was that of a man with an enormous abdominal tumour which nearly filled the abdomen. It was the size of a moderate oval-shaped football. It was soft and degenerating, and presented all the aspects of cancer or sarcoma. A piece was removed, and the microscope pronounced it round-celled sarcoma. The whole of that tumour has disappeared. The patient is quite well, and is a night-watchman in Cavendish Square, and I often see him as I go home in the evening. I say these cases are most remarkable, but such instances are not isolated. I bring them forward to show how important it is that we should, as

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surgeons and clinicians, have a more accurate classification of these varieties of tumour than we at present possess.

Passing now to the most important subject, in which you are all interested, cancer of the breast. The origin remains a great puzzle, and it is a thing which often strikes me, but I have not been able to elucidate the mystery, why cancer should occur in the breast and not occur in the parotid and submaxillary glands, because those glands are very similar in their component tissues, and there are ducts by means of which an infection could travel to the gland. Yet primary cancer of the submaxillary or parotid glands is very rare, notwithstanding that the structure is almost an exact counterpart of that in the breast. And it is very suggestive clinically, when we say that cells of the salivary glands at forty or fifty are not in the state of degeneration which they are in the breast, and therefore the ideas arise that cancer of the breast may be a parasitic disease which attacks degenerating cells, or the proper physiological functions of the cells being taken from them, they begin to undergo riotous proliferation. But, all this can hardly be the case in the liver, where cancer is so frequent. Another most suggestive point for us to think of is that, as a rule, an exact imitation of the primary growth is seen in the secondary growths. This is a very important matter, and I have been struck very much lately in examining a number of infected axillary glands in cancer of the breast to find how exactly the epithelium in the secondary infected gland simulates that in the primary growth. Some of the cancers of the breast originate in the ducts; then the epithelium is columnar. Take the instance so often quoted, cancer of the liver secondary to cancer of the intestine. Then the cancer of the liver consists of columnar epithelium. Consider the example where you get secondary growths after cancer of the thyroid gland in the bones of the skull. These are so exactly like thyroid gland itself, that it would puzzle microscopists to say whether a specimen were cancer of the bone or of the thyroid gland. If cancer be a parasitic disease, how is it that the secondary cancer cells so exactly simulate the cells of the tissue in which the primary growth originates unless it be an irritative parasite, which is carried in the cells, an intra-cellular organism. All these reflections are of the utmost importance. When one's mind is balanced between the cellular and the parasitic origin of cancer, I cannot help thinking that that matter of the secondary cells exactly simulating the primary cells of the growth is in favour of the cellular origin of the disease rather than its parasitic origin. Every impartial observer will allow that there is the closest relation between what I may call normal cell-proliferative growth and certain malignant tumours, especially the varieties of sarcomata. Let us take a common instance, and that is the reproduction of a limb in a crustacean. You know certain crustaceans have the power of shedding their limbs, and a new limb will be developed almost as perfect in its form and function as the original limb. If you have examined the early stages of reproduction of a limb in the crustacean you will be struck by the enormous amount of cells produced in an exceedingly short time. The amount of cells is much in excess of the immediate requirement, just as in sarcoma. And yet these cells are subsequently arranged in layers in regular order, and are developed into normal tissues, instead of into new growths! Going back earlier still, what is more remarkable than the phenomena which occur after the meeting of spermatozoa and ovarian cells. The cell growth which is manifest in a few days is extraordinary. The mass of cells which form is far greater in the time than in a new growth, and the structure is much the same—embryonic tissue. Yet the cells range themselves into laminæ, and they become complex in arrangement, and everything is arranged in a quite mysterious manner, which should make us ponder before we speak of our knowledge of the developmental faculties of cells. There is no parasite at work here! And the pregnant woman will frequently display anæmia and loss of flesh as the fœtus grows, a close mockery of the so-called cachexia which accompanies rapidly growing sarcoma! Our ignorance of the laws regarding cell proliferation and their multiplication is great. And you will all feel that my remarks concerning the cell origin of cancer must be a little disappointing. But we must recognise that the first state towards real knowledge is acknowledgment of ignorance. And an investigation of the processes of which we are ignorant is the first step to improvement. A knowledge, therefore, of the normal processes of cell growth is very needful, and I have an idea that the explanation of tumour growth is

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largely bound up in the mysteries of normal cell growth and of the extraordinary qualities of protoplasm, of which we know so little. This brings one to a conclusion which I would like to force upon the attention of this meeting to-night, and that is, the enormous need in this country, and indeed in all civilised countries, for proper investigation into this great subject of the origin of cancer: I mean the proper furnishing of laboratories, with proper, adequate, lasting, financial support. Here proper workers may investigate this great subject from the many points of view which I have barely indicated to-night, if needful, through a long series of years. Moreover—and here again the words have been somewhat taken out of my mouth by the remarks of Sir Mitchell Banks—it is of the greatest importance that the workers in these laboratories should be the right sort of men, men who work literally for the investigation of this subject rather than for their own glorification, and especially for anything which might promote their own interests, in puffing a so-called "cure." The financial aspect of the case is an all-important one, and skilled investigators must be adequately remunerated. Again, I may strongly support what he has said, that the most special care should be taken by investigators into this subject not to advance flimsy theories about the origin of cancer, or airy statements regarding remedial measures, which come before the public. There is nothing which undermines the influence of the medical profession with the public so much as inaccurate and careless statements about cures of incurable maladies which are afterwards found to be erroneous.

I will detain you only another moment while I speak of the enormous importance of what I call the correlation of clinical and pathological work. With regard to those numerous instances where cancer does not return, and is apparently cured, Mr. Gould has spoken to us of such. I have had a number of cases, I do not know how many, of patients who have had bad cancer of the breast removed, whose axillæ have been cleared out, and who are well a considerable time, four to five years, after the operation. I have one patient well eleven years, and another twelve years, after operation. Is this due to my skill in operating, or to my particular kind of operation? No, it is not. It is due to an undetermined factor in some individuals which militates against the return of the disease. I am sure there are certain persons who have a "something" which is inimical to cancer growth. And when we find long freedom from cancer after operation, such cases should be studied in all their aspects most carefully, and then possibly something will be discovered which will throw light on the subject. Again, I have a case which I have seen only to-day, a patient who has remained well for seven years after removal of cancer of the breast, and now has undoubted cancer in the glands of the neck. Primary cancer does not occur in glands of the neck. I believe the cancer elements must have remained in these glands all these years, and now have only just begun to show themselves. There must be something remarkable in this patient to prevent the growth having been developed before. I can support the remark of Sir Mitchell Banks, and say that the fortunate patients are generally thin, spare and withered people, they are not gross in habit, great eaters, vascular and full-blooded. Again, I know the case of a gentleman æt. 74, who had cancer of the floor of the mouth. I am sure Mr. Gould will agree with me when I say that a more rapidly fatal form of cancer does not exist than that of the floor of the mouth. That patient was seen by several eminent surgeons, and one said, "Do what you will, he will be dead in six months." He had masses of growth under his jaw on each side. Two operations were done. The cancer of the floor of the mouth was removed by one, and then all the glands were dissected out of the neck by an extensive "flap operation" at the second. It is now nearly three years since, and he remains perfectly well. I do not understand the case. I am certain that in ninety-nine out of a hundred such cases the patient would have been dead in six months, with glands enlarged, and reaching to the mediastinum. But he is an old, withered man, and his tissues appear practically starved.

Such joint work as I have indicated may really produce some useful results, results which may, in years to come, form justification for a meeting such as this. When more useful knowledge has been accumulated I believe we shall be in a better position to speak of it than we are now. I believe the explanation of tumour growth is very largely bound up in a greater knowledge of the normal processes which govern the development and proliferation of cells, and physiological as well as

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pathological research is urgently needed in the investigation of this, one of the leading secrets of nature, the greatest mystery that any man can be called upon to solve or investigate.

Dr. BARRY BLACKER.—Mr. President and Gentlemen, my remarks to-night are upon the present position of X rays and ultra-violet rays in the treatment of cancer. The success which has attended the employment of physical methods in the treatment of various skin diseases, more especially the somewhat tedious but satisfactory results obtained in the treatment of lupus, has been followed by the employment of the same or similar methods in the treatment of cancer, more especially of those varieties which are situated in the more superficial positions, and it is these methods that I propose to demonstrate to-night. These may be divided into three classes :

1. The light treatment associated with the name of Professor Finsen, and introduced by him.

2. The electric brush discharge from any high-tension electrical machine.

3. The X rays.

As light is only electric vibrations of short wave length, and an X ray, in the opinion of many, also electric vibrations of still shorter wave length, it is probable that there is no essential difference in the treatment. It is to a great extent a question of penetration, and the question is to find out which wave length is capable of penetrating the normal tissues without injury and producing effects on the deeper structures either by the destruction of bacteria contained within them, or by causing some alteration in abnormal less stable cells leading to their absorption. The physical data on which Professor Finsen's process is founded are (*a*) that the light from the sun and from an arc lamp is known to be rich in the most refrangible rays, *id est* the blue, violet, and ultra-violet, and that these are destructive to many forms of bacteria, (*b*) that the ultra-violet rays have but little penetrative power for most substances, the penetrative power increasing with the wave lengths, extremely short waves having scarcely any penetrating power at all. Short waves will penetrate quartz, water, gelatine, etc., fairly well, but are stopped by glass, mica, blood, etc. ; in order to penetrate the skin, therefore, it is necessary to render the part bloodless by compression. Air also offers an obstruction, and most of the ultra-violet rays given by the sun are absorbed by the atmosphere at ordinary levels. Owing to the difficulty experienced in producing rays of sufficient penetrative power, only the superficial forms of cancer can be treated by this method.

There are five forms of apparatus for the production of these light rays :

(1) In the first, the sun is used as a source of rays, the rays being filtered through a condenser which contains a fluid coloured blue, and then through a compressor in which water is circulating ; these absorb the heat rays, and the compressor renders the part treated bloodless. (Apparatus exhibited.)

In the remaining four the electric arc is employed.

(2) The original Finsen telescope condenser and compressor. (Apparatus exhibited.)

(3) The Lortet and Genoud lamp. (Apparatus exhibited.)

(4) The iron electrode lamp which is cooled by water. In this form of apparatus the employment of the water-cooled compressor is dispensed with. (Apparatus exhibited.)

(5) The condenser discharge lamp. The water-cooled compressor is also dispensed with, a block of ice being substituted. (Apparatus exhibited.)

The advantage of the three latter forms of apparatus is that the part to be exposed is brought much nearer to the source of the rays, all the condensing apparatus having been dismissed and only the compressing portion with its water supply or ice interposed in the path of the rays, by which the period of exposure has been shortened from one hour and a quarter to ten or fifteen minutes, and sometimes even to five. The compressors have also undergone many and great improvements. I show you all of those which are at the present time employed. These improvements have all been produced by gradually lessening the thickness of the media through which the rays have to pass. In the last form, which has been made for me by Messrs. Beck, the quartz has been ground down to extreme limit capable of bearing the pressure of the water, and on the surface of the patient without breaking, the water space for the circulating water is only 3 mm., and the part exposed is only separated from the arc by a distance of 2·2 cm.

In regard to the electrical brush discharge, as far as we are able at present to determine, it would seem as if this method of treatment presents much the same features as Finsen's. It is by Dr. Leduc, of Nantes, and Dr. Strebel, of Munich, that this method has been introduced. It differs, however, in the source from which the rays are derived. An electrical brush discharge is a powerful source of invisible ultra-violet, and visible violet, and blue rays, with a small admixture only of heat rays, for

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the chemical rays given off act like light on a photographic film; they are concentrated by a lens, and are reflected and concentrated by a concave mirror. They convert oxygen into ozone, and they set up fluorescense in barium platinocyanide and other salts; moreover they produce a reaction on the skin resembling that produced by the sun or an electric arc, which differs from the reaction following exposure to the X rays in being produced and healing quickly, instead of appearing slowly and healing slowly. The employment of the brush discharge has many advantages owing to the absence of heat rays and the presence of violet rays, without the admixture of all wave lengths. The most convenient apparatus for producing a manageable brush discharge is a Tesla or similar transformer (apparatus exhibited), and from these a number of patients can be treated at the same time. The object is to obtain a blue discharge from an electrode shaped so that pressure can be applied to the surface. For this purpose an ordinary test tube fitted with an ebonite sheath, leaving only the rounded end exposed, forms the best electrode (apparatus exhibited). The test-tube is filled with water and the end plugged with a rubber cork, through which a copper wire fitted with a terminal passes into the water. The terminal is connected with one pole only of a Tesla. The current passes down the tube through the water and is stopped by the glass, but, owing to the condenser effect, appears to pass through it and to reappear on side nearest to the patient's skin as violet light, accompanied by a small amount of heat which is absorbed by the water. Another form of electrode (apparatus exhibited) used by Dr. Leduc consists of a small metal sphere placed on one side of a quartz diaphragm, the quartz being enclosed in the same manner as the diaphragm of a telephone. In this form the glow proceeds from inside of the diaphragm as well as from outside, and as the quartz is permeable an increased effect is produced. This form cannot be cooled by water. Instead of the Tesla transformer the high frequency apparatus devised by Dr. Oudin, of Paris, may be used; it is called a resonator, because he makes use of the resonant effect of the current flowing in two parts of the same coil to increase the tension, instead of transforming up in another winding. It is possible that the electric brush discharge, apart from the violet rays proceeding from it, may be the active agent, and the matter is complicated by the ozone given off and by the products of electrolysis, which must be generated to some small extent in the skin from the decomposition of sodium chloride, etc.

I have left the description of the treatment by means of X rays to the last, as the physical data on which the treatment is founded differs so essentially from the foregoing methods; with all of them it is possible to destroy bacteria, but under the X rays many of them flourish; the same complications of ozone and electrolytic products, besides the brush discharge from the tube, are present as in the electric treatment, but the great penetrative power of these rays marks a wide distinction between them and the violet.

At first sight it does not seem probable that rays which pass through the body could be capable of producing effect, and it would seem more possible that effects might be produced by some of the rays which are absorbed. The well-known action on the skin produced by the X rays, and variously described, shows that alterations and destruction of cells may be produced, and the difference of the onset and the result from that obtained from the violet rays demonstrates that a difference exists in their action. The destructive action of the skin is usually produced by the action of tubes having a comparatively low vacuum, and giving off rays of low penetration, and yet the result of experience teaches us that this destructive action is not a necessary accompaniment of the process which results in the diminution and contraction of an ulcerated surface, or, in some instances, of the diminution in size of subcutaneous growths, for these results can be obtained equally well by a tube having a high vacuum, which only rarely produces destruction of the skin. If the destruction of the superficial tissues was necessary before a subcutaneous growth could be acted upon, it would be more convenient to use the salts of Radium and Polonium, etc., the effects of the rays given off by which have been studied by Professor Becquerel, Madame Curie, Herr Griezel, etc., for if the skin be exposed to the action of these salts for a few hours no result will be observed for about three weeks, but after that period an ulceration will be produced which will take another three weeks to heal; for instance, Professor Becquerel, by carrying an active salt in his pocket for six hours produced an inflammation reaction, which appeared ten days later and took thirty-nine days to heal.

In conclusion, it would appear that we have various rays which may be utilised in the treatment of cancer; we have violet rays which penetrate the tissues only to a small extent, and are bactericidal and produce some tissue change; we have the X rays, which penetrate the whole of the body, and produce tissue destruction, but are not bactericidal; and we have mixed rays, which can be produced by various methods, and it does not seem unreasonable to suppose that rays may be isolated which may be found capable of destroying bacilli or acting upon various cells in different parts of the body. X rays especially are known to ionize the air through which they pass, *id est* to break up the molecules. Theoretically, therefore, the field of research in this direction would seem very extensive.

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* *Specially reported for The Clinical Journal. Revised by the Author.*

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A CLINICAL LECTURE

ON

"SOME AFFECTIONS OF THE MYOCARDIUM."

Delivered at the Medical Graduates' College and Polyclinic.

By G. A. GIBSON, M.D., D.Sc., F.R.C.P.Edin.

GENTLEMEN,—In acceding to the very generous and flattering invitation of your Dean that I should come and lecture upon some subject here, I had the temerity to ask, after our northern fashion, whether a lecture on the myocardium would be acceptable. When I was told it would, I began to realise the difficult position in which I had placed myself, because on consideration I found the subject to be alarmingly extensive. From every point of view it is, in truth, large and wide. It is a trite observation to say that the heart, being connected with every part of the body, reacting to every stimulus, suffering under every influence, must necessarily have its ramifications most extensively distributed; therefore the field to be covered by any general lecture upon the myocardium is too large. It will probably conduce to your comfort, as it certainly will to my convenience, to pick out two or three aspects of disease of the myocardium for our special consideration. But before doing so there are certain points as regards the historical aspect of diseases of the muscular tissue of the heart that it will be well to pass in review.

The earliest physicians seem to have considered that the myocardium was practically exempt from disease altogether; and following upon this view, there were some amongst their successors who regarded the myocardium as incapable of suffering from any primary disease—from any idiopathic affection. In fact, until we come to the days of Cohnheim, there was probably no one who considered that the heart might suffer from protopathic lesions, that is to say, from primary affections.

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Acute myocarditis, or acute inflammation of the muscular tissue of the heart, was certainly observed by Morgagni,* but the microscopical structure of it was first elucidated, long afterwards, by Hamernjk,† of Prague; a correct understanding of such acute inflammation, however, only came through the observations of Craigie,‡ of Edinburgh, and the whole of this subject was systematised by the German pathologist Stein.§ Chronic myocarditis was likewise observed by Morgagni.‖ From two different points of view it was further studied: from the standpoint of toxic influences setting up chronic changes in the heart wall it was investigated by Lancereaux,¶ and as the result of chronic interference with the circulation it was observed by our own great countryman, the late Sir William Jenner.** Fatty degeneration of the myocardium was observed in the first place by Corvisart.†† He applied the teaching of Vicq d'Azyr‡‡ regarding the degeneration of muscular tissues in general to those of the heart in particular. But this subject did not receive systematic treatment until Sir Richard Quain§§ worked it out in that admirable paper which was published in the 'Medico-Chirurgical Reports.' Fatty accumulation or infiltration, a mere deposit of fat in the subpericardial and subendocardial tissues, leading by invasion often to subsequent fatty degeneration, was first observed by Harvey,‖‖ who describes it in his immortal work. It was further worked up by Bonetus,¶¶ and by Lancisi,***—not in his work upon the movements of the heart, but in his work on sudden death.

Of the secondary changes which ensue upon some of these, which we may almost call primary disease, we have dilatation and hypertrophy. Dilatation was certainly observed by Harvey,††† but it was fully studied by Willis.* Senac,† in his great and monumental work on the heart, fully dealt with the causes of dilatation for the first time, and Morgagni‡ almost put the copestone upon this edifice by finding out what some of its effects were. It is interesting to notice that Mayow§ was the first to describe hypertrophy. He described hypertrophy of the left auricle in cases of mitral disease, while Vieussens‖ observed hypertrophy of the left ventricle in cases of aortic disease. The subject of cardiac hypertrophy received academic treatment at the hands of Bertin,¶ whose notions, however, although they dominated the pathology of the heart for well-nigh half a century, have been finally disposed of, largely by the efforts of Cruveilhier.** Atrophy of the heart was also observed by some of the ancient writers, particularly by Riolan.†† Senac‡‡ also made some observations upon this subject, and Allan Burns,§§ of Glasgow, contributed very largely to what we now know of simple atrophy or wasting of the heart. This also was systematised by Bertin‖‖ in his work on the heart.

Such is a very brief synopsis of the development of what we know as to the pathology of the myocardium. From the point of view of clinical medicine, Auenbrugger, in his great work,¶¶ was certainly the first to notice alterations in the size of the heart, and, as is known by every one, Laennec *** formulated, from a far more developed standpoint, principles and methods of making out differences in the size and in the force of the heart.

The first of the two subjects which I wish more particularly to bring before you is the intimate relationship of the myocardium to the vascular supply of the heart. It will probably be regarded as an observation altogether trite to say that the

* 'De Sedibus et Causis Morborum,' 1762, i, p. 291.
† 'Ost. med. Jahrb.,' 1840, S. 1 und 139.
‡ 'Edin. Med. and Surg. Journ.,' 1848, lxix, p. 86.
§ 'Myocarditis,' 1861, p. 70.
‖ *Loc. cit.*
¶ 'Traité de Pathologie,' 1879 to 1881, ii, p. 767.
** 'Med.-Chir. Trans.,' 1860, p. 199.
†† 'Essai sur les Maladies du Cœur, 1806, p. 185.
‡‡ 'Œuvres,' 1805, v, p. 365.
§§ 'Med.-Chir. Trans.,' 1850, p. 121.
‖‖ 'Works' (Syd. Soc.), 1847, p. 590.
¶¶ 'Sepulchretum,' 1700, i, p. 888.
*** 'De Subitaneis Mortibus,' 1707, p. 176.
††† *Op. cit.*, p. 128.

* 'Opera omnia,' 1682, p. 103.
† 'Du Cœur,' ii, p. 393.
‡ 'De Sedibus et Causis Morborum,' 1762, ii, p. 24.
§ 'Tractatus,' 1674, p. 96.
‖ 'Traité,' 1715, p. 102.
¶ 'Traité du Cœur,' 1824, p. 283.
** 'Traité d'Anatomie pathologique,' 1856, iii, p. 13.
†† 'Encheiridion,' 1649, p. 219.
‡‡ *Op. cit.*, ii, p. 393.
§§ 'Observations,' 1809, p. 110.
‖‖ *Op. cit.*, p. 387.
¶¶ 'Inventum Novum,' 1761, p. 88.
*** 'De l'Auscultation médiate,' 1819, iii, p. 267.

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possibilities of nourishment of the heart depend, firstly, upon the general condition of the blood; and secondly, upon the limitations of supply by means of the coronary arteries. It is to be remembered that there is something almost bizarre in the apparent distribution of these coronary vessels. The left coronary artery happens to be smaller than the right, and at first sight this would almost seem as if Nature were at fault in giving a smaller vessel to the left side of the heart. But the apparent discrepancy is thoroughly explained by tracing out the whole distribution of the coronary vessels, since the left coronary artery is found to supply little except the anterior wall of the left ventricle and a small portion of the adjacent anterior wall of the right ventricle. The right coronary artery supplies almost the entire right ventricle, the whole of the auricles, and the posterior aspect of the left ventricle; hence its larger supply. We cannot but think that there is a possibility thus generously provided by Nature for evading disaster to the systemic circulation if anything should happen to one of these coronary vessels. And let me say in parenthesis now, that to one who has been long engaged in studying vascular changes in the heart it is very interesting to find that the left coronary artery may sometimes be almost entirely invalidated—may be absolutely insufficient to carry out any nutritive work, and yet the heart is able, through the patency of the right coronary artery, to go on doing its work fairly well. Amongst the workers who must be held in honour for observations on the relations of these vessels to the myocardium there stands first in order Bellini, the great Italian pathologist. Bellini * in the seventeenth century observed changes in the coronary arteries, changes which, in those old days, were summed up as ossification, but which we now know to be simply arterio-sclerosis going on to its ultimate result in calcification. Sir William Gairdner † in 1854—the year in which Stokes's immortal work on the heart was written—promulgated some very interesting views as to the connection between the coronary arteries and the heart wall. He did not, however, pursue the subject further, but left it as a suggestion, and it was a considerable time before the seed thus sown may be regarded as having borne fruit. Weigert,* in his work on coagulation processes, for the first time really elaborated the theory that when there is interference with the blood-supply to the muscular substance of the heart, there must be some of the changes which I shall try to bring before you more fully afterwards. He was immediately succeeded by Charlton Turner,† who, just one year afterwards, at the meeting, which we can well remember, of the International Medical Congress in this city, produced a very interesting paper on this subject. Huber,‡ a German writer, followed closely upon the track of these observers, and the theory might well be accounted as thoroughly worked up. But a few years later, in 1887, Lindsay Steven,§ of Glasgow, went over all the field once more, and found that the circulation in the heart is really of the nature of a terminal blood-supply, meaning thereby that its connection with the circulation is, as in the brain and most of the internal organs, by means of arteries going straight to their destinations, and passing into their arterioles; these go directly through the capillaries into the venules, by which the blood comes back with little or no anastomosis. Therefore, changes in the arterial distribution in the heart must necessarily produce something analogous to what we know as softening of the brain. We must give full credit to Thurnam ‖ for his observations upon one aspect of this subject. He led us to recognise the intimate connection which takes place between such patches of softening due to vascular interference and cardiac aneurysm, or to bulging of the wall of the heart. Nor should I like to pass away from this without also referring to the wonderfully clear work of Zeigler ¶ upon acute heart softening or myomalacia cordis.

If we try to look into this subject a little more closely, we shall find that when there is a total sudden occlusion of one of the great coronary arteries there is invariably sudden death. The heart at once strikes work, as was pointed out by

* 'De Urinis et Pulsibus,' 1685, p. 615.

† 'Monthly Journal of Medical Science,' 1854, p. 79.

* Virchow's 'Archiv,' 1880, p. 106.

† 'Transactions of the International Medical Congress,' 1881, i, p. 427.

‡ Virchow's 'Archiv,' 1882, p 236.

§ 'Lancet,' 1887, 1153, *et seq.*

‖ 'Med.-Chir. Trans.,' 1838, p. 187.

¶ 'Lehrbuch,' 1881, i, p. 408.

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Erichsen,* giving the same result as the tying of a coronary artery, and the changes which are seen on post-mortem examination are practically the same in both cases. There is no alteration in the tissue; it is simply anæmic, there having been no time allowed for any definite changes. This, then, we may regard as a result of a total and sudden occlusion. It is most common to find this produced by embolism, and I observe that my friend Osler,† in his work on medicine, speaks of coronary embolism as being rather frequent. It is far from being so in my own experience; and coronary aneurysm, also, although it does take place, is certain one of the rarer cardiac lesions.

Much more often seen than this total and sudden occlusion is a complete but gradual blocking of one of the coronary arteries. There is under one of the microscopes a very beautiful instance of this which was under my observation some years ago. It is from a patient who came into the ward under my charge with every appearance of cardiac failure. There was nothing to lead to a definite diagnosis. It was quite true that he had incompetence of his great venous valves, both mitral and tricuspid, but there was nothing to show us that these were primary, and the natural conclusion, therefore, was that he was suffering from some form of myocardial breakdown, very probably due to some vascular changes. He had some arterio-sclerosis, as shown in the radial and temporal arteries, but he had low blood-pressure. The result of these facts was to lead us to conclude that the heart wall had broken down in consequence of some vascular change. After lingering for some time, partially recovering, and then relapsing, then again to some extent regaining strength, he finally died, and it was found that the whole of the left ventricle, upon its anterior wall, was of exactly the same thickness as the right ventricle—one eighth of an inch,—that the muscle fibres were entirely replaced by newly formed connective tissue, and if you will do me the favour of looking at the microscopic specimen you will see that in the part of the section which is in the field there are only three or four bundles of muscle fibres, all the rest are replaced by actively growing fibrous tissue.

A partial sudden occlusion leads to myomalacia cordis, that change which has been so thoroughly described by Ziegler.* The common changes leading to these are old-standing sclerosis of a coronary artery and a gradual process of thrombosis which eventuates in a sudden change, because, although a blood-vessel of very small calibre will keep up a certain amount of heart function, still a time comes when these cannot be maintained, and the patient dies suddenly. On the other hand, this may take place from a sudden thrombosis; the section under the second microscope shows such a condition as this. In this myomalacia of the heart wall it is found that there is a very rapid fading away of the striation of the heart muscle, a considerable number of red blood-corpuscles may be seen amongst these muscular fibres, and, if the death has not been very sudden, the commencement of a fibrous process, all of which are particularly well seen in that microscopic slide of myomalacia cordis.

When there is a gradual partial occlusion due to sclerosis of vessels, leading to a very gradual thrombotic process, in addition to these changes, there are simply patches of fibrous tissue; we may say that in looking at sections of the muscle wall in such a condition as this, there seem to be little islets of more or less altered muscle tissue with the striation somewhat disappearing, and with a granular appearance throughout, in the midst of a whole sea of fibrous tissue, which is, for the most part, very thoroughly nucleated. The third microscope shows such changes wonderfully well.

Now the interesting point for us as physicians in all these effects of coronary disease is this: that when the heart has undergone such alterations as these, it necessarily has a tendency to fail when there comes to be any period of storm and stress, and this is, I believe, the correct explanation of the fact that pneumonia, as well as all the other acute infections, takes on such a very serious aspect after middle life. The last three microscopes show cases of this kind. The first is from a case of interstitial myocarditis due to malarial infection, in which the patient died somewhat suddenly of what, under other circumstances, would probably have been a transient acute affection. The second shows a somewhat similar change due to arterio-sclerosis, in which the patient died, so far as the surgeon could tell, quite unaccountably after an operation for strangulated hernia, from which, in

* 'Lond. Med. Gaz.,' 1842, p. 561.

† 'Practice of Medicine,' 1892, p. 641.

* *Loc. cit.*

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his view, the patient ought to have recovered. The third shows what is in the common experience of most of us the cause of death in a case of acute pneumonia, in which you will see also that there is arterial sclerosis, which has led to a chronic interstitial myocarditis.

From the point of view of diagnosis of these conditions, which is for all of us as physicians their most valuable aspect, it must necessarily be difficult to formulate any hard and fast rules. But it is common to find that the patient begins to suffer from symptoms of cardiac failure; the balance of the circulation is, be it ever so little, somewhat disturbed; there is a tendency to breathlessness on the least exertion, a liability to crepitations at the bases of the lungs; and if along with such premonitory symptoms, which may be regarded almost like danger-signals, we find there is a little puffiness about the ankles, that the urine has a tendency to be scanty and high-coloured, or that there is a trace of albumin, we can tell that something has destroyed the integrity of the myocardium, and the rest of the diagnosis must necessarily proceed by the method of exclusion. Given healthy blood-vessels, I mean given radial arteries which appear still youthful, with temporal arteries which are not tortuous and which cannot be distinctly felt, we may find it rather difficult in the absence of valvular lesion, in the absence of any proof of an adherent pericardium, to make up our minds as to what is causing this change in the heart. But if we find that there are these vascular changes, or if we find that the patient has had attacks of acute infections, we may be led thereby to the conclusion that there is some myocardial change. But having come to such a first conclusion, how are we to say that it is a myocarditis? Can it not just as well be a fatty degeneration? Now fatty degeneration, according to some of our authors, is an affection very easy to diagnose. According to other and wiser authorities, it is a matter of the greatest difficulty to determine. Probably we shall all be at one in believing that when there is for a considerable length of time any accumulation of adipose tissue in excess throughout the system at large, the heart will suffer. The healthy heart, as we know from post-mortem examination, has a comparatively small amount of fatty tissue, chiefly deposited in the sulci, and, to a slight extent, along the distribution of the blood-vessels; when there is a tendency, such as I am afraid all of us who have passed middle life have, to a little expansion of our waist,—in short, to a fatty deposit, no matter how slight, the heart will necessarily suffer. And if this accumulation goes a step further, to form what I think we may well call encroachment or infiltration, there may be a third effect produced, there may be degeneration of the muscle fibres of the heart itself, from the pressure upon them of this invasion.

Now how are we to make up our minds when there is such a fatty change as this? It is said by some of our authors, glibly enough, that if we find a patient with the arcus senilis, with a liability to corpulence, a tendency to breathlessness, and so forth—and I have even seen mentioned, in otherwise sober and sane works, a somewhat greasy skin,—that we are then at liberty to diagnose fatty accumulation and infiltration. Gentlemen, I think nothing can be further from the truth. The arcus senilis has no more to do with fatty degeneration of the heart than the moon has to do with green cheese; they are as far apart from each other as the poles. The arcus senilis must be at once put out of court as having the least utility from a diagnostic standpoint in these conditions. Similarly we must not base too much upon the mere fact of whether the patient has a slight tendency to corpulence or not; but here we are probably on safer ground than when considering the arcus senilis, because when there is accumulation of fat in any part of the body the heart necessarily shares in that. In making these remarks it must be clearly understood that I do not at present refer to the fatty degeneration which occurs as a result of certain toxic processes, or which takes place as a part of pernicious anæmia, and many of the acute infections. These changes may be suspected, and the suspicion is only too often found to be true at the subsequent post-mortem examination, but that suspicion has nothing to do with the diagnosis of a fatty degeneration occurring in the way I have indicated, from a too great deposit of fat upon the surface of the body.

Of other degenerations nothing need be said, because they are relatively uncommon. Hyaline degeneration, occurring as it does in diphtheria, in pneumonia, and in many other infections, is only part and parcel of a vitreous degeneration of

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muscles throughout, and I need not waste a single sentence upon this aspect of the subject.

Likewise I need say nothing about pigmentary atrophy, which is essentially a senile process simply taking place as one of the retrogressive changes in elderly persons; or about waxy degeneration, which is one of the most uncommon of the cardiac affections. Therefore, amongst the common changes of the heart we have chronic myocarditis on the one hand, and fatty changes on the other; there is, however, an almost intermediate condition, because the two very frequently go hand in hand—interstitial myocarditis is very frequently accompanied by granular changes. These are nothing but the commencement of a fatty degeneration, and it is, perhaps, safest for us when we are dealing with a patient who has arterial sclerosis, and also a tendency to the deposition of fat, to conclude that there is some chronic interstitial fibrous change along with some fatty degeneration.

From the point of view of prognosis little need be said. These chronic interstitial changes necessarily lead to grave disaster whenever the heart muscle is exposed to any serious task, and, as has already been said, speaking pathologically, in such conditions when a patient suffers from an acute infectious disease, or when he is exposed to great worry in the turmoil of life, he very frequently passes away from cardiac failure, simply in consequence of the fact that the heart cannot further be nourished. And it must be in the experience of every one of us that in those startling cases, in which life comes to a termination with a suddenness which is quite dramatic in aortic disease—where incompetence is the most salient feature of the case—the end of life arrives simply because the nutritive possibilities of the coronary arteries are inadequate to sustain its functions any further. When we find there is a probability that the heart wall has undergone some degenerative process our prognosis for the patient is necessarily a somewhat grave one.

As regards treatment, it would hardly become me, addressing an audience of my fellow-practitioners, to say much with regard to this aspect of the subject; but I may confine myself to a few generalities. In addition to trying to keep the patient in as thorough general hygienic surroundings as possible, we should study every aspect of his environment most sedulously. When we turn to drugs we find there is none which is of such supreme importance as one or other of the iodides: iodide of potassium or iodide of sodium—both of these will be found invaluable. Along with this there should be thorough care in regard to regulation of all the excretions, and the use of general hæmatinics and tonics, more especially iron and arsenic. If such are insufficient, then there may be added to them strychnine, and, last of all, it may become necessary to add strophanthus or digitalis. There is probably not one among you who cannot recall many patients in and beyond middle life in whom vitality is simply kept up to what we may call a condition of adequacy by the use, on the one hand, of drugs such as the iodides, and, on the other hand, by the use of cardiac tonics, of which I believe strophanthus to be far and away the best.

The other aspect of the subject which I wish to bring before you is to try to answer the question, Is there such a thing as any primary myocardial disease? Probably there is scarcely such a condition as primary disease of the myocardium. Threatened on every side, as it may be, by all kinds of noxious influences, it may yet maintain wonderful integrity; it is probable that there is but one primary affection of the myocardium which we may say is inborn with the individual, and which we may call myocardial insufficiency. No doubt we all remember how the late Sir Andrew Clark,* some years ago, brought before us an old friend with a new face when he christened an inability on the part of the kidneys to do their duties thoroughly as "renal inadequacy." A man is often born with a stomach which is insufficient for the average duties it is called upon to perform. We know, on the authority of Murchison,† that a man is sometimes born with a liver which is not quite equal to, at any rate, excessive calls upon it. We recognise that many a man is born with brains which are scarcely sufficient to see him through life; the same thing is necessarily true of the heart.

The question of cardiac insufficiency or inadequacy frequently comes before me, because education in our northern capital being one of the staple products, youngsters are frequently brought to me suffering from this affection in varying degrees. They

* 'Lancet,' 1879, vol. ii, p. 800.

† 'Diseases of the Liver,' 2nd ed., 1877, p. 613.

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can get through life on what we may call a low platform, but whenever the exigencies of life become serious, when life becomes intense, there is at once a failure to meet the demands, and what has been so well called overstrain of the heart is one of the effects. I can at once recall several instances of that kind brought to see me by various friends of mine. One very interesting youth, who had been training for his school races, was brought with a considerably dilated heart, and with mitral and tricuspid systolic murmurs. A few days of absolute rest, with no cardiac tonics at all, simply with sedulous attention to food and rest, at once restored that youngster to comparative health; and by hard and fast rules to avoid strain to the heart in the future he has, during the last two or three years, never had the least tendency to a relapse at all. Another similar case which at once rises before my mind was that of a hard-working undergraduate going up for one of his legal examinations. In the brief hours that he allowed himself for relaxation,—he was one of those youths who worked until the sun almost rose on the following morning,—in those brief hours he allowed for relaxation, he spent in chasing a golf ball over one of the most hilly links around Edinburgh. The result in his case was a total breakdown of the heart. By giving that youth perfect rest for a time, and then by putting him upon a regular system of gentle exercises along with general tonics, not heart tonics at all, he gradually became a strong and healthy man. I happened yesterday, when I was driving along one of our streets, to catch sight of him coming down the old High Street of Edinburgh looking as ruddy and as healthy as need be. In addition to such forms of strain, one other was brought before me the other day. I was asked to go out to one of the great schools near Edinburgh with the medical attendant of that school to see a boy of about sixteen years of age. He had come from South America not very many months before, and he had begun to play football. He was a big, strong, healthy-looking youngster. He played hard as a forward, and on one of his practice days, when he was just in the act of getting the ball, he happened to stumble and roll round upon his back, and another boy of about his own size came heavily upon his præcordium with one of his knees. At once he became in a collapsed condition and was troubled with a good deal of cardiac pain, radiating in the typical manner of angina pectoris. He had every symptom of cardiac failure and a notable degree of cardiac dilatation. In his case rest, with but little medical treatment, brought him back to a satisfactory condition; but I had a note from his doctor a few days ago to say that on attempting again to take some active exertion, he had for the second time broken down, and had to be sent to bed.

Now what I wish to bring before you, lastly, to-day, gentlemen, is the question, What is the mechanism by which this cardiac failure takes place? It is, no doubt, simply that the myocardium is not quite adequate to the stress that is placed upon it. Stress begets strain, and dilatation ensues. But of what kind is this dilatation? We know well, from the interesting observations of Roy and Adami,* and from the very admirable anatomical researches of Hesse † and Krehl ‡ that the heart during diastole has a considerable amount of dilatation of the muscle rings surrounding the mitral and tricuspid orifices, and we may call the muscle tissue surrounding these orifices the mitral and tricuspid sphincters. From a series of casts obtained by Hesse during systole and diastole we may say that this point has been proved to demonstration. In addition to a general dilatation of the ventricles in conditions of stress, it seems to me extremely probable that we have a further relaxation of the sphincter surrounding these orifices, whereby there is allowed a free regurgitation in many of such cases of cardiac strain backwards into the auricles. I am glad to say that just a few months ago Morton Prince, of Philadelphia, has been working at this subject, and in the 'American Journal of Medical Sciences' § he has published a very interesting paper dealing almost entirely with the clinical aspect of such cases. Some observations he has given have struck me as being singularly true to Nature. We all know, especially those of us who are sometimes called upon to examine for life assurance, that patients come under a condition of what we can only call considerable mental perturbation, and as

* Brit. Med. Journ.,' 1888, ii, p. 1321.

† 'Archiv für Anatomie und Entwickelungsgeschichte,' 1880, p. 328.

‡ 'Abhandlungen der Königlichen sächsischen Gesellschaft der Wissenschaft,' xvii, p. 339.

§ February, 1901.

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one of the results of this the heart acts far more powerfully, and sometimes in those applicants—we cannot call them patients—there is developed not merely a tricuspid systolic murmur, but even a mitral systolic murmur. It seems very probable that in such cases we have to deal simply with what we may call a physiological or safety-valve action of the sphincter, not only of the tricuspid but of the mitral orifice, that, in short, the safety-valve function which Wilkinson King, of Guy's Hospital, brought forward * may be applied also to the left side of the heart, but not equally so. Added to this we must remember that a dilatation of the chambers of the heart will lead to a maladaptation of the cusps on account of the musculi papillares and chordæ tendineæ not being able to adjust themselves perfectly to the new conditions, and thereby a certain amount of escape is allowed. The importance of this conception in regard to diagnosis cannot be over-estimated. We all know how fallacious it is to form a diagnosis, and still more to frame a prognosis upon a cardiac murmur, and when we find that some of these cardiac murmurs that meet us every day of our lives may be regarded as almost, if not quite physiological, we shall certainly avoid falling into many an uncomfortable pitfall. How are we, then, to diagnose such conditions? We can only diagnose them by finding that there is, first of all, no valvular disease, *i. e.* by deciding, both from the history of the case and from the evidences that are before us, that a primary valvular lesion may be, to all intents and purposes, safely excluded. When we have done that, and when we make certain that the patient does not labour under any chronic myocardial degenerative change, we may then have a certain degree of confidence in predicting that there is a purely transient affection, and that the outlook is satisfactory.

As regards the treatment of such conditions, we may sum the matter up in the one term "rest." Rest is the best of all the cardiac tonics, and in such conditions as these it is of paramount importance; but, added to rest, there should be a thoroughly adequate, yet not too stimulating diet; regular exercise, as soon as possible, which will help to oxygenate the patient's blood; avoidance of everything which will make for arterial degeneration; avoidance of too highly nitrogenous food, of all beverages which have a tendency to lead to arterial sclerosis, of all overstrain which will lead to dilatation; all these points must receive our most careful attention. In addition to these there can be no doubt that we can, especially when dealing with youth, do a very great deal to develop the muscle of the heart and to lead it to withstand strain when it is subjected to stress, because by means of graduated exercises, whether resistance or otherwise, more particularly I believe by exercises which are useful for the respiratory apparatus, we can very greatly help to lead such patients to grow up in the future to the enjoyment of a healthy life.

* 'Guy's Hospital Reports,' 1837, ii, p.104.

ON THE SPONTANEOUS DISAPPEARANCE OF GENERALISED CANCER.

By J. BLAND-SUTTON, F.R.C.S.

In the issue of this Journal for May 28th, p. 96, Mr. Pearce Gould refers to a case under his care at the Middlesex Hospital with recurrent cancer two years and a half after removal of the breast. Whilst under observation the cancer nodules disappeared, the ascites absorbed, the liver diminished in size, and the patient improved so markedly that she was discharged from the cancer wards at her own request. Mr. Pearce Gould has omitted a very important feature in the clinical history of this patient. In February, 1899, I removed the breast from this woman (æt. 41) and the axillary glands. Five months later she came under the care of Dr. Hugh Fenton on account of a large pelvic swelling, which proved to be due to distended pus-containing Fallopian tubes; these sacs, with both ovaries, he removed in June, 1899. Dr. Hugh Fenton saw the patient in the middle of May, 1902, and confirms Mr. Pearce Gould's statements completely in regard to the disappearance of the recurrent nodules and the shrinking of the liver, but there was a discharging sinus in the scar of the abdominal incision. In the light of recent knowledge this disappearance of the cancerous nodules cannot be called "spontaneous," but has some relation to the double oöphorectomy. Even from this point of view the case is a remarkable one because the subcutaneous nodules, the enlarged liver and hydroperitoneum made their appearance in a woman whose ovaries had been removed, and then slowly subsided.

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WITH

MR. MALCOLM MORRIS

AT THE MEDICAL GRADUATES' COLLEGE AND POLYCLINIC.

I HAVE asked this gentleman to come here to-day so that you might see him. He is a private patient of mine, and has been good enough to sacrifice his own feelings for the public good. He is forty-six years of age, and he has suffered from skin trouble ever since early childhood; in fact, with the exception of very short intermissions, which I will refer to more in detail in a moment, he has practically never been free from irritation through the whole time. He can remember very well suffering severely when he was a boy. The trouble is everywhere about him, but more especially on the extensor surfaces. The skin is thickened and harsh and rough as a result of these years of scratching. The itching is not always present in the same degree; sometimes he is comparatively free from it, while at other times he has fearful paroxysms. He has noticed that the itching is worst when he is hot. If you look closely into the skin, I think you will be puzzled to find a primary lesion anywhere; as a matter of fact, there is very little to be seen except as a result of the scratching. This is a type of skin disease which we see more especially in the upper classes; we only occasionally see these cases in the lower classes, and I have only seen three or four at the hospital. You can feel how thick, harsh, and rough the skin is as a result of all this scratching. No one would call it an eczema, except by using the term in the most casual way. It is a case of Hebra's prurigo. He is forty years of age, and has taken every care of himself, which he is in a position to do. In addition to that he has been suffering ever since he was sixteen years of age from severe spasmodic asthma. There are certain places which he cannot live in. He lives in the country, where he has estates, and when he goes there has fearful attacks of asthma, during the occurrence of which, however, his skin condition undergoes some amelioration. At the age of fourteen he entered the navy, and the doctor who examined him for the navy said he was suffering from scabies, and that he must be put back for the cure of it. He had been suffering ever since he was a child from the disease, but it was not scabies, which, indeed, he never had. The remedies which he had gave him temporary relief at that time. In addition to his asthma he has a lump on his wrist-joint. One of the diagnoses which have been made is that he has got gout, that he also has eczema, and that there is also gouty asthma. Now I venture to put a different interpretation on the group. I believe he has got rheumatoid arthritis, an asthma which is a pure genuine neurosis, and Hebra's prurigo—a combination which is fairly common. The cases are not common, but the combination of these symptoms is not uncommon in such cases when they are met with. The exact diagnosis is extremely important, because if this is a genuine gout, and he is suffering from a gouty condition of the skin and mucous membrane—that is to say, if his asthma was also gouty in origin—he needs an anti-gout line of treatment. He has been through a considerable number of treatments from this point of view. Recently, he has been to Harrogate, and has gone through a sharp course of treatment there *qua* gout. But he came back very much worse than he had been previously, showing that that kind of treatment was not particularly successful. What treatment is likely to have any beneficial effect on the condition? As to internal treatment, what drug is likely to have the effect of stopping the three neuroses which I have mentioned? We must admit it is an extremely difficult thing to decide what remedies will control these three neuroses. But the drugs which I have seen produce a very considerable amount of benefit in many instances of like character are valerianate of zinc, cannabis indica, hyoscyamus, and that class of remedy. He is taking valerianate of zinc and hyoscyamus in a pill. I have never seen any benefit in such cases from the administration of arsenic. Naturally, if a person has suffered from a chronic skin affection from childhood onwards and lives to this age, it is a certainty that someone will have administered arsenic, but my experience is that arsenic rather aggravates the symptoms. The class of remedy I have mentioned is only with the view of altering the man's general nervous condition so as to modify the phenomena which are dependent upon the nervous system. Of course, other drugs must be given to relieve the actual symptoms. So far

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as the asthma is concerned, he burns papers in his room, but he has tried everything to relieve this spasmodic asthma. With regard to the condition of the skin it is very difficult to find any drug or combination of drugs, either in the form of lotions, or baths, or ointments, which will give any real relief. You may, perhaps, think that a pretty active course of tar applications might modify the condition; but he would still tear himself in his sleep, and there would be the thickening of the skin persisting. There is one very simple remedy which he is using, and which I think he will admit he is getting relief from, namely, simple carron oil, with which he thoroughly lubricates the skin. I should expect more relief from internal remedies than from local. Of course, warm baths take away the itching for the time.

(A member of the class said it was believed that tartarised antimony, $\frac{1}{16}$ to $\frac{1}{8}$ grain, three times a day would relieve the irritation in a very marked manner in dermatitis.)

I wrote one of the original papers, in 1878, on the use of antimony in skin affections, and I have the greatest possible belief in it, but in this particular combination of diseases I should be a little doubtful as to its efficacy, because there is an element in the case which I will speak of when the patient has left. He has never suffered from mosquito bites; indeed, he says if he was in a mosquito country with a mate, the mosquitoes would seem to go to his companion instead of to him. I must now tell you that the patient's father died in a lunatic asylum, where he lived for thirty years prior to his death. He has brothers, two of whom are in asylums at the present time. So I do not think there is any doubt about neurosis in the case. This patient has had extremely bad attacks of melancholia, and that is one reason why I should be a little afraid to give antimony. I am prepared to admit the value of antimony in general dermatitis, but this is a purely neurotic condition. There is very little the matter with his skin if he did not continue scratching it as he does. The way he behaves when these paroxysms come on is extraordinary; he will tear off his clothes, and appear to be irresponsible for his actions. The paroxysm may last for an hour or half an hour. For a long time his two conditions alternated; that is to say, at one time his asthma would be worse, and at another time his skin.

Here is a man, about thirty-six years of age, who has had both palms of his hands affected for three or four months. He says he has never felt anything the matter with his throat, nor had any eruption, though he admits that he had something on his penis last June. To have the first eruption in syphilis on the palms is certainly unusual. This may be an early secondary eruption. There is not the slightest sign of enlargement of glands in his groin. The man now seems to be in perfect health. I have given him perchloride of mercury to take, and I expect to see the condition get well. I have given him a mercurial ointment to rub in. Both his palms have been exposed to the same amount of friction. There is a fissure evident in the uvula, which looks like an old matter. He says he has not had any sore throat or quinsy. Both his hands became bad at the same time.

Here is a man, about fifty years of age, whom I showed here last July, when he had a very bad syphilide. You will see the result in very bad scarring. He had a gumma on his penis and much œdema. He has been taking iodide of potassium continuously ever since that time, and you will see how great the benefit has been. I bring him again so that you may see the effect of treatment. He has not had any mercury. He has had fifteen grains of iodide three times a day.

Here is a boy who presents a rare case of disease, namely cystic tumours with colloid degeneration. I showed him once before, in the summer, on the first day he came to me. I now show him again after we have had an opportunity of making an investigation. He is a pathological curiosity. I ask you not to press the parts much, as he gets purpura on the slightest pressure. You see these tumours, sections from which are under the microscope for your inspection. If you touch them lightly with the fingers you will find that they all move freely under the subcutaneous tissue. At the time we first saw him we did not know what the tumours were. They are cystic tumours with a dense fibrous capsule, and with apparently a colloid degeneration in the centre. That is about all we can make out from microscopical examination. They have been examined by a colleague of mine, and that is the report of their nature. In addition to these congenital tumours in various parts of the body, he has displacement of the bones at the elbow-joint due to an old fracture, and a

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purpuric condition of the skin. He has also what is known as elastic skin. Every now and then some of these tumours suppurate, and you will see one of them now suppurating. The case is nearly unique. In that wonderful book which has lately been published by Alexis Thompson, of Edinburgh, there is a remarkable series of pictures of fibromata, some of which seem to look like this, but there is nothing in the book exactly like it. The condition is very much better since he has been in the hospital. He is an exceedingly clever boy, and is very active. He has congenital heart disease, probably mitral trouble. The tumours are movable, but they have moorings so that their oscillations are limited. When squeezed they are painful. One of the theories which have been advanced is that there have been hæmorrhages under the skin, either in uterine life, or at all events while he was an infant, which have become organised and so formed these tumours. Of course it was a mere theory. He gets bad hæmorrhages. With regard to elastic skin, I have a specimen taken from the skin of the elastic-skinned man at Barnum's show, who could hold the skin of his chest up and look through it. When this man found he had this peculiarity he cultivated it. I have searched through literature for a similar case to the present one, but have not been able to find one.

CANCER: ITS ORIGIN, NATURE, AND GENERAL PRINCIPLES OF TREATMENT.

(*Debate continued from page* 112.)

Mr. D'Arcy Power.—Sir, I wish, in the first place, to offer you my thanks for allowing me to come here this evening, although I am not a member of the Chelsea Clinical Society, and I should at the same time like to express my pleasure at the way the framers of the discussion have put this question, because it is one which allows a very large subject to be approached on all sides. Although I am a surgeon, I came here to-night to speak rather from a scientific point of view, because if we can find a cause for cancer, we shall be very likely indeed to find a cure for it. Like many other people, I began some years ago to work at the microscopical appearances of cancer, and more and more I have been dissatisfied with the results which have been obtained by this method, because, I think, they have led to nothing so far, except to a better knowledge of epithelial cells and their staining properties, and of certain blastomycetes or fungi.

Lately, I have been trying to work out the cancer question on somewhat different lines. I thought it was possible that there might be some intermediate host in connection with cancer, for cancer is found more abundantly in some parts of the country than in others, and those parts of the country where it is most rife are, broadly, as Mr. Haviland has pointed out, low-lying places, where a considerable number of trees are present, where the water is sluggish, or the soil is actually water-logged.

Thus, when casting about for a cause of cancer, I have thought that possibly flies might serve as intermediate hosts. Flies are accountable for the spread of many other diseases. For instance, Dr. Tooth, when he was in the Portland Civilian Hospital in South Africa, said that flies were, no doubt, carriers of enteric fever. At the Modder River, where there were a large number of cases of sunstroke and enteric fever lying in a tent side by side, on looking into the tents one could distinguish the people who had sunstroke from those who had enteric by the number of flies which were attacking the enteric patients. No sooner did an enteric patient put out his tongue, than flies settled upon it in abundance. Then, of course, it has been known for a long time that tubercle and cholera have been carried by flies, for the bacilli have been found on their feet and in their excreta.

The way I set to work was as follows: I made myself thoroughly acquainted with the anatomy of the ordinary house fly, by microscopical sections, and so forth. When this was done, I got a friend, who was living in a district in which cancer was very abundant, to catch me some flies in a house where there had been a succession of cases of cancer. The particular house was one in which a farmer had died four years ago from cancer of the rectum. The whole family moved out of the house, and new tenants came in. This family consisted of a father, a mother, a son, and a daughter. The first thing that happened was that the daughter, a woman æt. 36, had cancer of the

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uterus, of which she died. Whilst my friend was treating her for cancer of the uterus, the father asked him one day to treat him for piles. When he came to examine for the cause of the piles, he found that the father was suffering from a carcinoma of the rectum. Things have gone from bad to worse. He has had colotomy done, and my friend says he is now in a very bad way. Since the father has been ill, the mother has got some trouble with her antrum, and my friend thinks it is probably sarcoma or epithelial disease of the upper jaw. I thought, therefore, that this was a favourable house to get flies from. I examined, hardened, and stained sections of these flies very carefully, and I may say at once that there is nothing at all in the flies from the rooms of that house which is in any way different from the appearances seen in sections of flies taken at the same time of year from a house in London where there had been no cases of cancer.

This result, I think, is rather what one would expect. In the first place, an ordinary house fly is not an insect which has any power of penetrating the human epithelium. The head has suckers, but no cutting apparatus, and the ovipositor has so many foliaceous appendages attached to it that there is no possibility of its penetrating human epithelium. I think, if you are going to find a micro-organism in connection with flies, you should look for a true inoculation to take place by some means of penetration.

This important point, however, came out of my examination of flies, viz., that in late autumn it is quite common for flies to bite, and they bite very sharply. There is no question about it; they have the power of penetrating the human epidermis, and they will raise a little spot at the site of the injury. There is not much reaction around it in healthy people, but still there is a definite bite. My out-patient department at St. Bartholomew's Hospital shows me every year that there are many people who have been bitten by flies with the most unpleasant results. Within the last year alone I have had three people under my care in whom the bite of a fly has produced a carbuncle, the patients having distinctly attributed the carbuncle to the injury produced by a fly, in each case during the months of August and September. I could not understand how flies came to bite from time to time, so I asked my friend Dr. Lowne, who published recently an important monograph on the blow-fly, and he solved the difficulty at once. He said, if you look about, you will see that there are really two kinds of flies; there is the Stomoxys calcitrans, which is comparatively rare, and there is the ordinary house fly. You can distinguish the one from the other quite easily, because the stomoxys is smaller than the house fly, and keeps its head erect in the air. On its back it has three little white interrupted bands, which do not exist in the house fly. If you examine the mouth-parts of the stomoxys, you will see he has a complete set of lancets, and that he is capable of biting, and does bite, in the late autumn. That biting fly is always the stomoxys, and never the common house fly. Yet these two kinds commonly live together, and are to be found in the same places. He said he had often found them in Bayswater and Kensington, when he was practising in London, so they are no strangers to town life. On the other hand, they occur typically and most numerously in places where there are cattle, and in low-lying districts.

It is for that reason I have come here this evening. I want those who are interested in the question of the origin of cancer to ascertain as far as they can whether the flies in any particular cancer house are of the genus stomoxys, that is to say, biting flies, or whether they are the ordinary house flies. It may be rather difficult at first to distinguish the one from the other; but by watching and seeing the way in which they hold their heads when they are at rest, one should have little difficulty in the matter. Besides that, I should be very glad if anybody would send me living specimens of flies from a cancer house, particularly the stomoxys. I do not care about the house fly, because I know he has nothing inside him except what he ought to have. The third point which I think one might inquire into—I do not know whether it is worth anything—is to find out if people who are suffering from cancer can remember whether they have been bitten by flies in the preceding autumn. The bite is so slight and transient that I do not suppose anybody would be very much troubled about it at the time. But it is possible that information on those three points might bring about some definite knowledge.

To sum up, therefore, what I have said, I want to ascertain, by sections, whether inside a given fly

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there are any of the many appearances which have been called "cancer bodies," or whether there is any indication whatever of the fly being an intermediate host, in the sense that it has in its tissues appearances which one cannot explain by the ordinary histological or pathological changes. One knows perfectly well the various forms of degeneration to which epithelial cells are liable, and one knows some of the different changes that flies undergo in process of development. These developmental changes, however, make the subject difficult to work out, because the fly undergoes very radical changes from time to time in the course of a year. Each insect goes through a series of developmental changes, in which its tissues become extremely modified. The few positive results I obtained I have sent to the *Edinburgh Medical Journal*, where, I hope, they will be published in due course, with two or three drawings. The appearances which are there represented can be explained in other ways than by thinking we have an intermediate host to deal with. But this line of experiment has not been very much worked at in connection with cancer, and I should be glad of any facts or any flies which could be sent to me by those who are interested in the subject.

The following communication was then read to the meeting by the Chairman :

Note on the Presence of Cancer in Swine.

By Harvey R. Gaylord, M.D., and
C. H. Zink, D.V.S.
From the New York State Cancer Laboratory, University of Buffalo.

The recent experimental work of various observers dealing with the etiology of cancer has more than ever attracted attention to the distribution of this affection. It is of special interest to ascertain the distribution of this disease among animals. The most recent article dealing with the subject is that of J. M. McFadyean in the 'Practitioner,' April, 1899. After giving a list of forty-nine cases of cancer in animals, including the horse, dog, ox, cat, and sheep, McFadyean makes the following observation :—"It will be observed that only one of the common domesticated species is not represented in the total : it is the pig ; and the writer has never met with a case of either carcinoma or adenoma in that animal. There appears to be no room for doubt that cancer is very rare in the pig."

For the purpose of ascertaining to what extent carcinoma might be found in swine, the assistance of Dr. C. H. Zink, Director of the Local Bureau of Animal Industry, was obtained. About 25,000 head of swine per year are slaughtered in the Dold institution in Buffalo, under the inspection of Dr. Zink. In a period of less than two months, Dr. Zink succeeded in detecting four cases, which, on examination, proved to be malignant growth in swine.

The tumours consist, first, of Case 68, liver of pig containing multiple metastatic nodules. In section, stained with hæmatoxylin-eosin, these nodules present the characteristics of a typical, soft, infiltrating carcinoma. The primary seat of the carcinoma in this case, as in the others, with the exception of the kidney cases, was not obtained.

Case 70, brought in by Dr. Zink December 20th, 1899, is enlarged kidney, weight 1620 grammes. The superficial aspect of the kidney is about equally divided between red cortex and a white, irregularly nodular mass, involving at least two-thirds of the cortical surface of the remainder. On section, the tumour is found to be of distinct lobulated character, of whitish-yellow colour, about the same consistence as the cortex of the organ. Very little of the cortical substance remains, and consists of a compressed layer of cortex spread out upon the tumour, and represents the remains of the inferior cortical portion of the organ. The tumour mass is supported by a well-defined connective tissue stroma, enclosing irregularly shaped cavities lined with multiple layers of cylindrical epithelium. Into the larger openings project papillary structures covered with the same epithelium. In many portions of the tumour the regular arrangement of the epithelium disappears, and the cells are found scattered in more or less well-defined nests in the stroma. Karyokinetic figures are infrequent. Diagnosis, papillary adeno-carcinoma of the kidney.

Case 70A. Kidney of swine. The organ is enlarged. About one half of it is involved in a whitish, firm mass, which extends the capsule. On section it is found to be of a whitish colour, with a consistence somewhat less than that of the kidney cortex. The kidney cortex is atrophic, about one-half remaining. Section stained with hæma-

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toxylin-eosin. The tumour is composed of connective tissue, the cells of which are generally of the spindle variety and vary greatly in size. Certain portions of the tumour contain a large amount of inter-cellular substance, the nuclei in these areas being smaller than in the rapidly growing portions of the tumour. Certain fields of the tumour present the characteristics of spindle-celled sarcoma, the nuclei showing great variability in size, and staining deeply. Karyokinetic figures are very rare. Scattered between the spindle cells are occasional small round cells. Diagnosis, spindle-celled sarcoma of the kidney.

Case 72. January 3rd, 1900. Large abdominal tumour of female pig. Weight of carcass alone, without tumour, 113 pounds; weight of tumour alone, 58 pounds. Tumour appears to consist of a mass of enormously enlarged lymph nodes. These, on section, are whitish in appearance, with occasional areas of hæmorrhage. The masses are somewhat softer than hyperplastic lymph nodes. A few metastatic nodules in the lung. Microscopic section stained with hæmatoxylin-eosin. On section, the characteristic structure of the lymph nodes has disappeared, its place being taken by a complicated structure of cells with a delicate vascular connective tissue stroma. Under high power the cells are found to possess deeply staining, spherical, or oval nuclei, surrounded by a cell body with a deeply staining protoplasm. In some cases the cell body is so small as to be scarcely apparent. The arrangement of the tumour is in many cases of definite adenomatous type, the cells being arranged in single layers around spherical, oval, or irregular openings. Groups of these gland-like structures are apparently formed into lobules. In many cases they appear to rest directly upon open spaces which contain red blood corpuscles, or coagulated albuminous material. In these cases the cells appear to rest directly upon the endothelium lining the blood-vessels. In many portions of the tumour the cells are closely packed and present no definite structure, but in all cases they present an appearance which suggests their derivation from pre-existing lymph spaces. Owing to the arrangement of the cells and their character, the diagnosis of endothelioma is made.

The detection of four tumours, undoubtedly of malignant type, although taken from a large material, about 2000 hogs, would seem to be sufficient to show that malignant tumours are not as rare as had previously been supposed. It must be remembered that these swine included many young animals, and all were supposedly in healthy condition when brought to the slaughter-house. It is, therefore, probable that in hogs of more advanced age, especially among those falling sick, which would be killed without ascertaining the nature of their complaint, a still greater frequency of malignant tumours might be noted. The supposed immunity of hogs to carcinoma has likewise been referred to by Dr. Braithwaite in a recent article.

Professor TANNER HEWLETT.—Mr. President and Gentlemen, I fear that I cannot add much to the valuable theories and facts which have been brought before the Society by previous speakers in this debate. Many points which I might have referred to have already been anticipated, and I therefore need detain you but a short time with any remarks of mine. With regard to the theories advanced to account for the development (not origin and nature) of cancer, Dr. Beatson's theory seems to me but another way of saying that "something" must cause the growth of a cancer, but of that "something" we are profoundly ignorant. I am not sure whether I quite understand the meat theory of Sir Mitchell Banks. If he suggests that the germ of cancer is conveyed by meat, then we should expect that the carnivora and the great meat-eating races, such as the Esquimaux, would be more subject to cancer than the herbivora, or the races that eat little meat; but such I think is not the case. Nor is there an excessive cancer mortality among butchers. But if meat *qua* meat is the suggested cause of the development of cancer, then the increased use of meat, which Sir Mitchell Banks says is coincident with the increase in cancer, may resolve itself into a question of over-nutrition, an intake of food in excess of that required for the repair of the natural physiological waste. Such an excessive intake might well be supposed in certain cases to lead to over-stimulation of cells, with consequent excessive proliferation, especially in parts in which the cells had been to a certain extent damaged, and their normal physiological activities consequently impaired. It would be interesting to know whether the affluent suffer from the incidence of cancer to a greater extent than the indigent.

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According to Newsholme ('Practitioner,' April, 1899) taking the comparative mortality figure of cancer for different occupations (*i. e.* number of deaths from cancer in 1000 deaths) as 44 among all occupied males between twenty-five and sixty-five years of age, clergymen are represented by the comparative mortality figure 35, lawyers by 60, doctors 43, commercial travellers 63, malsters 61, brewers 70, inn-keepers 53 (in London 70), inn and hotel servants 65, butchers 57, merchant seamen 60, but fishermen 46, coal miners 36, but chimney-sweeps 156; an almost inexplicable series of contrasts. Incidentally it may be mentioned that Newsholme, who is no mean statistician, considers that the increase in cancer is more apparent than real.

Of the prevalence of cancer in certain districts, especially in damp, clayey soils, near rivers which overflow their banks, it is difficult to give an explanation. There is one factor which such a condition of environment favours, viz. the mosquito, but there is not the slightest evidence at present to show that the mosquito conveys cancer.

The non-cancer areas seem to coincide generally with the districts which we should describe as the more healthy ones, and where the inhabitants lead an outdoor life, and are not blest with too much of this world's good things. The geographical distribution of cancer, in my opinion, merits renewed study. With regard to the salt theory of cancer of Dr. Braithwaite, who has adduced the Jews as a race who eat little salt, and among whom the incidence of cancer is low, it may, I think, be doubted whether the Jews do consume little salt. If salt be the cause of the development of cancer, then sailors of all people should suffer largely from the disease. They consume salt pork almost daily, and breathe and swallow salt in the air and spray continually. As already mentioned, the comparative mortality figure for merchant seamen is 60, for fishermen 46; these figures do not support the salt-diet theory. I have also looked up the records of the hospitals of the Seamen's Society for the four years 1897—1900 inclusive. I find during that period there were 459 deaths from disease, of which thirty-one were due to malignant disease; this gives a comparative mortality figure of 67·5 nearly. Of the thirty-one cases of death from malignant disease, one was due to rodent ulcer, five to sarcoma, and twenty-five to cancer or "malignant disease." Taking the figures for carcinoma and rodent ulcer only, twenty-six deaths, the comparative mortality figure works out to 56·6 nearly. If deaths from accident be included (twelve in the four years for adult sea-going males) the comparative mortality figures come to 65·8 and 55·2 respectively. These figures may be taken to represent the mortality from malignant disease and carcinoma among merchant seamen fairly accurately, I think, and are not so very different from those of Somerset House, and are directly opposed to the salt-diet theory.

The influence of irritation as a cause of the development of cancer can hardly be doubted, yet in twenty-five cases of carcinoma in the horse McFadyean points out ('Practitioner,' *loc. cit.*) that in no case did the neoplasm start from a place subjected to much irritation, *e.g.* the sites for the collar, saddle-girths, and crupper. McFadyean, moreover, comments on the infrequency of cancer of the tongue, uterus, and mammary gland in animals. Of all places where it might be expected that cancer would occur, if some of the current views be correct, the udder of the cow would be the most likely, yet it seems to be unknown in this situation. McFadyean also remarks that he has never seen a case of carcinoma in the pig. This will add to the interest of Dr. Gaylord's paper, which has been read.

Now to come to the theories of the origin and nature of malignant disease. In the first place, it is to be noted that there is no absolute line of demarcation between the non-malignant and the malignant new formations.

The metastases, which are the dominant characteristic of carcinoma and sarcoma, are completely simulated by the metastases, which occur occasionally in chondromata and lipomata.

The parasitic theory of the nature of carcinoma is a fascinating one, but to my mind is very far from proved at present. And in this connection I venture to express the opinion that the analogies which have been drawn between infective diseases caused by bacteria or protozoa, such as tubercle and coccidial disease, are far from being true analogies. In tubercle, for example, the infective agent, the tubercle bacillus, introduced at a certain point produces cell proliferation, the tubercle nodule, followed by necrosis, with toxæmia due to the absorption of chemical products; here we have the primary focus of the disease. Frequently secondary foci of the disease follow, simulating the metastases of a malignant growth. But if we inquire how the secondary foci are formed the analogy ceases. The tubercle bacillus in some way is transferred to the site of the secondary focus, and there grows and multiplies, and produces proliferation of the cells there present; the cells which proliferate are not carried with the parasite, but are constituent cells of the tissue in which the parasite happens to settle. In carcinoma, on the other hand, cells from the original growth are carried to the site of the secondary focus, and there grow and multiply, and form the metastasis, the cells of the tissue itself being only

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secondarily affected by irritation, pressure, etc., and taking little or no part in the formation of the secondary growths. So it is with the coccidial disease; the secondary growths are caused by the coccidium, which wanders, and produces cell proliferation of the part in which it settles, and not by cells carried from the site of the original growth.

With regard to the hypothetical parasite, the idea that it is protozoon in nature seems to be largely abandoned at present. Many of the Ruffer's bodies, which were at one time considered to be the parasitic organisms, are now, I believe, generally regarded as being of the nature of vacuoles, and it is interesting to me to recall that I suggested this view of the nature of these bodies as far back as 1895. Gaylord seems to be the chief exponent of the protozoon theory at present, but it is to be noted that his work has come in for some pretty severe criticism at the hands of Sanfelice in a recent paper in the 'Centralblatt. für Bakteriologie.'

When the parasitic nature of the structures stated to be parasites is called in question, the exponents of the parasitic theory retort that the critic has mistaken certain other structures, cell-inclusions, vacuoles, etc., for the true parasitic bodies, as witness the criticism of Borrel, and Sanfelice's reply thereto. But if you will look at the plates illustrating Plimmer's article in the 'Practitioner,' and at those illustrating Sanfelice's paper recently published, the structures designated parasites by these two authors appears to differ materially. The exponents of the parasitic theory are not wholly agreed as to what structures are and what are not to be regarded as parasitic. The reports of the cultivation of the parasite and of the production of cancer experimentally with the pure cultures, seem to me to be also unsatisfactory and inconclusive. Probably the best proof will be furnished when a parasite, blastomycete, or whatever it is, is cultivated from a typical carcinoma of some lower animal, and with the pure cultures the disease produced in a healthy individual of the same species. For it is very likely that the parasite producing carcinoma in man, if there be one, is different from that producing carcinoma in a rat, for example; and that the parasite producing carcinoma in man, although actually cultivated, would therefore very probably fail to produce carcinoma in a rat. From the fact that carcinoma cannot be inoculated upon animals of a different species it is highly improbable that cultures, if obtained, will produce the disease, except in animals of the same species as that from which they were obtained. In dealing with the supposed production of cancer by inoculation of cancerous material, or with cultures therefrom, the possibility of the coincident development of cancer in the experimental animal must not be lost sight of. In this connection Dr. Lambert Lack's experiment may be quoted. Lack formulated the theory that carcinoma was due to misplaced epithelium. In order to test his theory he performed an experiment at the Jenner Institute which I had the good fortune to witness throughout, and for the authenticity of which I can vouch. He opened the abdomen of a female rabbit, scraped the ovaries so as to destroy them, and allowed the scrapings to fall into the abdominal cavity. The abdomen was closed, and the rabbit kept under observation. Some eight months afterwards the animal began to emaciate and to suffer from respiratory distress, and on being killed masses of growth, microscopically an adeno-carcinoma, were found in the peritoneum, mesentery, liver, surface of the diaphragm, and mediastinum. Here an animal is experimented upon, a carcinoma of the expected type develops within about the right time, yet all attempts to reproduce this experiment have failed. Either some unknown concomitant factor induced the formation of the carcinoma as the result of the procedures employed, or, as seems more likely, Dr. Lack was the victim of one of the most extraordinary coincidences that has fallen to the lot of any experimenter, viz. the natural and spontaneous development of carcinoma in an animal that had been operated upon with a view to producing the disease. Such an instance as this may well make us ponder when we hear of the successful inoculation of cancer or of cancer cultures. Remembering how infrequent such positive results are, the coincident development of cancer in the experimental animals is a possibility, apart from the procedures employed. Failing the parasitic theory of cancer, is there any other explanation to be offered for the genesis of malignant growths? Personally I incline to the view suggested by Mr. Marmaduke Sheild, that the mystery of cancer is bound up in the mystery of cell proliferation in general. In uterine life, cell proliferation in the embryo must proceed at an extraordinary rate; what causes the cessation of this rapid rate of proliferation? And in the adult, what controls and regulates the necessary normal, regular proliferation required for physiological repair? Something must; but I fear that any attempt to unravel this something would lead to the region of wild theorising so much descried, and justly so, by previous speakers in this debate. For ideas in this direction I would refer my hearers to a thoughtful and suggestive paper by Adami in the 'British Medical Journal' for March 16th, 1901. Possibly in his communication to-night we may hear more of his views. In conclusion I do not wish to be misunderstood; to my mind the parasitic theory of cancer is not proven, but no one would welcome it more gladly than I, should the requisite proof be forthcoming, as I cannot help feeling that there is greater hope for the discovery of a remedy for this fell disease if it be parasitic than if it be non-parasitic.

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** Specially reported for the Clinical Journal. Revised by the Author.*

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A CLINICAL LECTURE

ON

ACUTE OSTEOMYELITIS AS A CAUSE OF HIP-JOINT DISEASE.

Delivered at the Medical Graduates' College and Polyclinic, February 26th, 1902.

By RUSHTON PARKER, M.B., B.S., F.R.C.S.,

Professor of Surgery in University College, Liverpool; Surgeon to the Liverpool Royal Infirmary; President of the Liverpool Medical Institution.

GENTLEMEN,—The cases to which I ask your attention are three in number only, but by them my subject will be sufficiently exemplified, occurring as they did at long intervals during the last twenty-seven years.

These three cases I have selected from among others because they have proved conspicuous landmarks in the progress of my own knowledge, and although there is nothing commonplace about any one of them, they may also serve as texts for much that relates to hip disease of a more ordinary kind. As a cause of hip-joint disease tubercle, of course, is predominant, and there are other causes, such as rheumatism, acute and chronic, occasionally pyæmia, and gonorrhœal rheumatism. There is even simple traumatic inflammation, occurring in severe sprain of a healthy joint, or in the too early resumption of use after dislocation and its replacement. But the mechanical treatment required for all is the same.

As I shall have to refer to Thomas's splints it will be well to commence with a few lantern slides to represent them.

Fig. 1 is a Thomas's hip splint, of the double variety, and one of the old-fashioned kind, formerly made with a strap crossing the front.

Fig. 2 is a single Thomas's hip splint, the form in which it has now been made for many years. The upright part of the splint is made of iron rod strong enough not to yield to movements of

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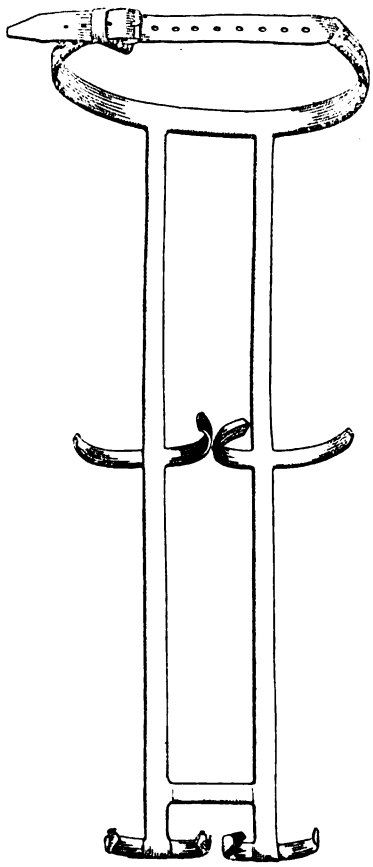
the patient, varying in width from an inch to an inch and a half, a quarter of an inch in thickness (sometimes it is only three-sixteenths of an inch thick for quite small and young people), and covered with a strip of felt as a padding, and all closed in with basil leather.

They can be made in a much cheaper form without any padding, and varnished or covered with tin-foil, the patient being then suitably padded with cotton wadding.

FIG. 1.

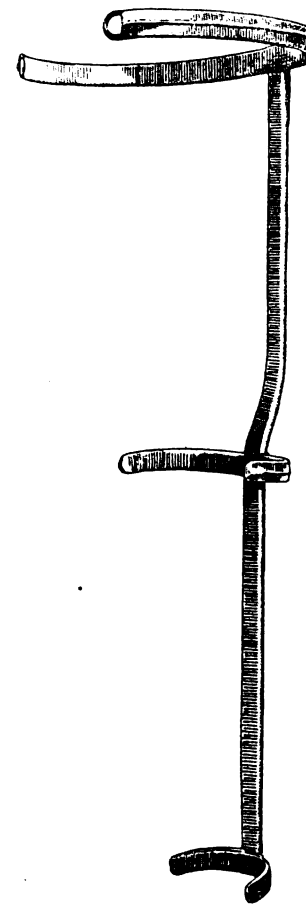


Figs. 3 and 4 show the splint fitted to a patient in the erect posture, front and back.

The next photograph is that of a little boy fitted with a similar splint lying down. This is a mode of applying the splint which I have used many times during the last twenty-five years. The boy had flexion of the hip-joint to the extent which you can see here (Fig. 5). A pillow was placed under the knee, and to maintain rest bandaged with him to the splint in the first instance without any attempt to disturb his flexed position. He was at once comfortable and at rest. At intervals of a few days the bandage was taken off, the chaff pillow pressed thinner, and the knee allowed to drop a little, "taking in the slack" each time, till the knee came conveniently down straight upon the splint.

The next photograph (Fig. 6) is one of the same boy before treatment, standing up as well as he can

FIG. 2.



against a table. The case is one of sub-acute tubercular hip-joint disease, and shows about the angle to which the hip-joint was flexed before treatment had been adopted. The next (Fig. 7) is the same patient lying in bed, or rather lying on the table and holding the limb with the left hand. I had that photograph taken to show the very characteristic attitude adopted sometimes by patients who have much pain in the hip-joint. Every patient with painful hip-joint disease is occupied during his

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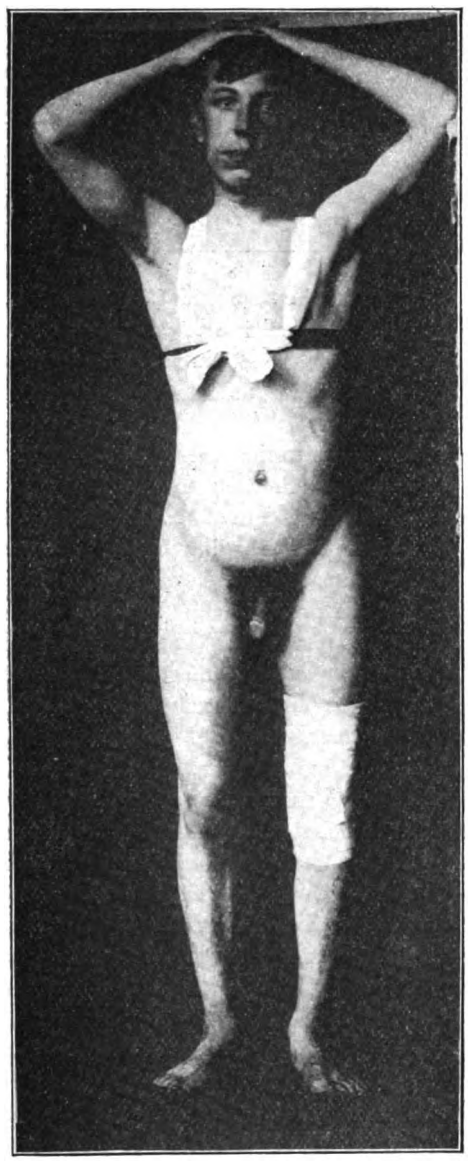
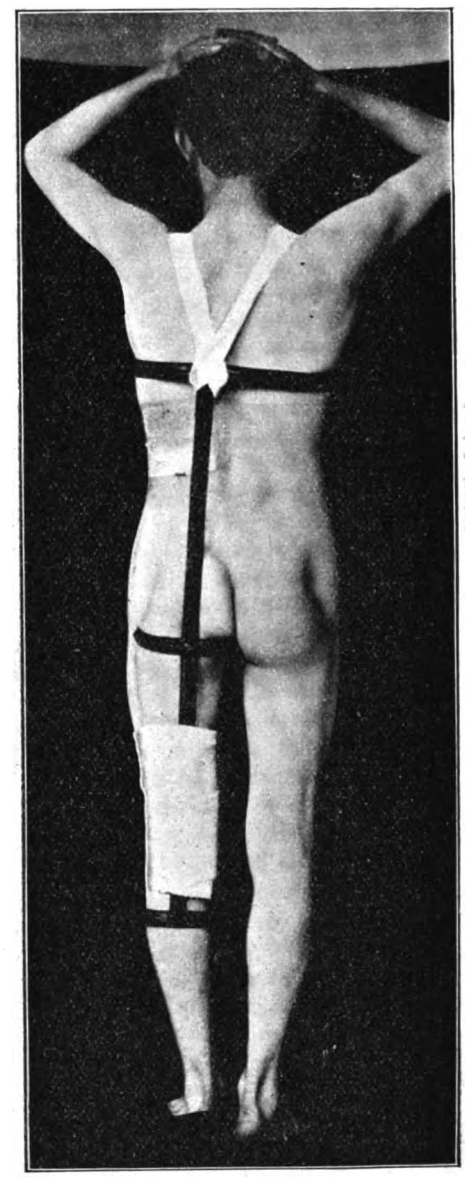
waking hours in keeping that limb still. The relief produced by a splint is due to the fact that the patient is relieved from the necessity of himself keeping the joint still, for all the muscles of the affected limb, and sometimes even of the

Fig. 3.



hand, are employed in holding the diseased limb still. When the patient goes to sleep the muscles relax, the joint moves and hurts him, waking him with "a start." Starting pains, in my opinion, signify that the limb is not fixed enough to prevent movements in the joint by muscular action. The attitude represented here is one which this patient frequently adopted, and I got him to assume it for the photograph. It is one which is well known in certain cases of hip disease, and I have

Fig. 4.



often seen other patients adopt it. I also show a photograph (Fig. 8) of the same boy with the sound knee bent up on the body, indicating the degree of flexion found in the hip-joint. The adoption of this attitude is a device of the late Mr. Hugh

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Owen Thomas for testing the presence of hip-joint disease as indicated by flexion, which is the only invariable sign in untreated inflammation of the joint. After the joint has been treated by mechanical fixation, the flexion may go in two or three days.

Fig. 5.

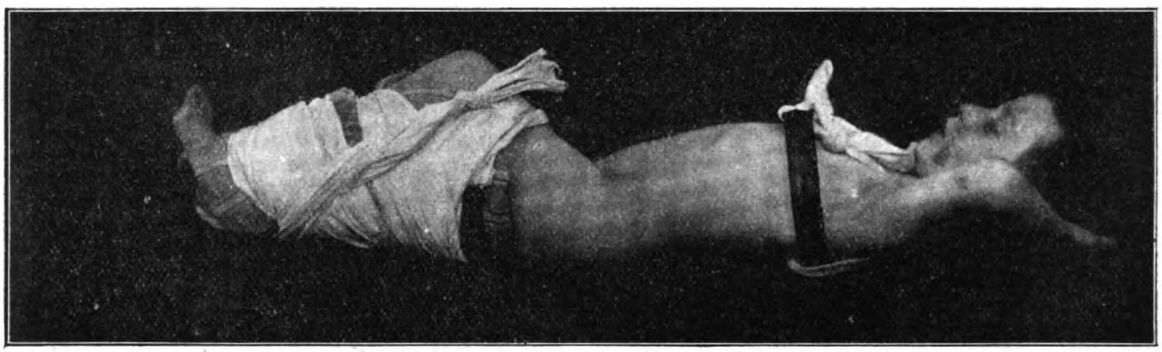
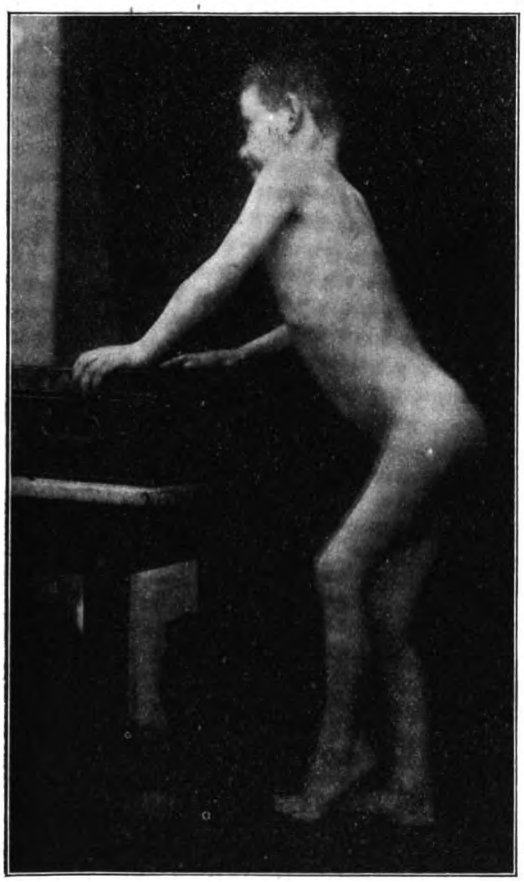


Fig. 6.



In addition to flexion there may be abduction, adduction, or rotation. The next (Fig. 9) is a photograph of the late Mr. Thomas himself, demonstrating his flexion test on a boy. The next (Fig. 10) photograph shows the flexion in another way, with the lower limbs straight and the back arched up.

I now pass on to the cases. In the first, a girl, æt. 8 years, was admitted to the Liverpool Royal Infirmary under the care of Mr. Edward Bickersteth on May 6th, 1875, having had, it was said, an attack of rheumatic fever eleven weeks before, after which a more or less acute inflammation of the right hip-joint remained. The left tibia and left humerus, moreover, were affected with small superficial abscesses and subsequent exfoliations of necrosed fragments of bone. At the time I supposed that the hip affection, in which I was keenly interested, was due to acute rheumatism, and did not at first perceive the entire significance of the bone affection in the humerus and tibia. A subsequent review of the circumstances, however, has led me to conclude that we had here a case of acute or sub-acute multiple osteo-myelitis and periostitis, affecting also the upper end of the femur and implicating the hip-joint. The early symptoms of this disease bear a close resemblance to those of rheumatic fever, and I have several times since that date operated on cases of the kind, for instance, cases where the tibia alone was affected, previously admitted into a medical ward as rheumatic fever. At the hospital in question, where I then held the position of pathologist, being also surgeon to the Stanley Hospital, I saw this patient with Mr. Bickersteth, who drew my attention to the case on May 23rd, 1875. We then

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found great flexion and adduction of the right lower limb, some rotation in, an apparent shortening of three or four inches, much flattening of the buttock, and an extraordinary looseness of the hip-joint under manipulation, but no pain, and only moderate tenderness on gentle handling. The extreme faultiness of the attitude in which the joint was found, together with the ease with which the limb could be placed in a straight position by manipulation, the entire helplessness of the patient to control the position of the limb which, when left to itself, at once relapsed into conspicuous deformity, lying for rest across its fellow; the looseness of attachment between the femur and the hip-bone permitting this "pathological dislocation," as it may be termed, and the obvious need of mechanical means to keep the limb at rest in line with the trunk, were all features of striking import. I had recently become acquainted with the late Mr. Hugh Owen Thomas and his splints, and as a result of the interview above referred to, Mr. Bickersteth accepted my offer to fit the patient with Thomas's hip splint, and placed her for that purpose entirely under my direction. Up to a short time previously the only trustworthy device known to me for such cases had been Liston's long splint, and I have never ceased to appreciate what can be done with that splint when carefully and efficiently applied. But in hip-joint disease its value is very limited, and since that period I have abandoned it for all purposes except that of instructing my class of practical surgery, in favour of the splints of the late Mr. Thomas. Mr. Thomas was then engaged in writing an account of his own splints and preparing explanatory illustrations for publication.* His splints have a value that has received

FIG. 7.

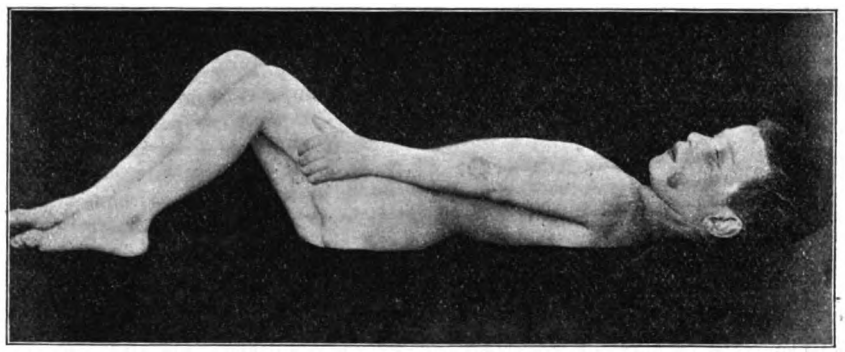
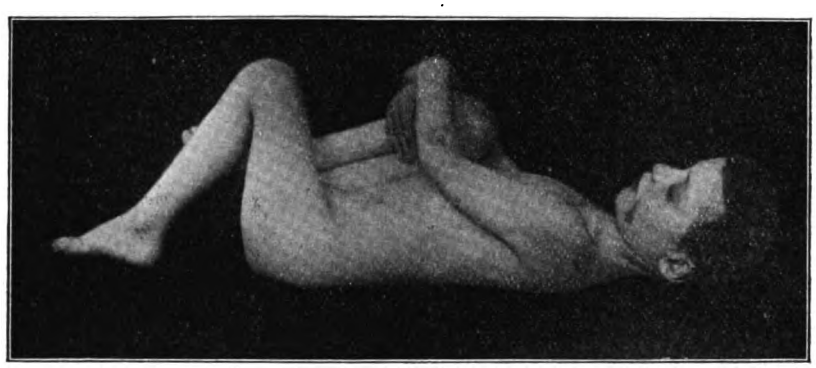


FIG. 8.



* Figs. 9 and 10 are copies of original photographs made for Mr. Thomas' work (1875) and printed then from drawings. They are here published for the first time in their photographic form, having been placed at my disposal by his relative Mr. Robert Jones. See also 'International Text-book of Surgery' (Gould and Warren), vol. i, 1900, p. 729.

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wide acknowledgment in many parts of the world, and I was intimately associated with him in making them known. This case has great historical interest for me, being the first in which Thomas's hip splint was used in the Liverpool Royal Infirmary, and one of the earliest in which I had myself used that instrument. Great interest was manifested and valuable assistance rendered by the house surgeon, Dr. W. T. Hayward, now of Norwood, South Australia, during the eventful and unfamiliar incidents that we met with. In order to procure a splint I had to superintend its construction by a

FIG. 9.

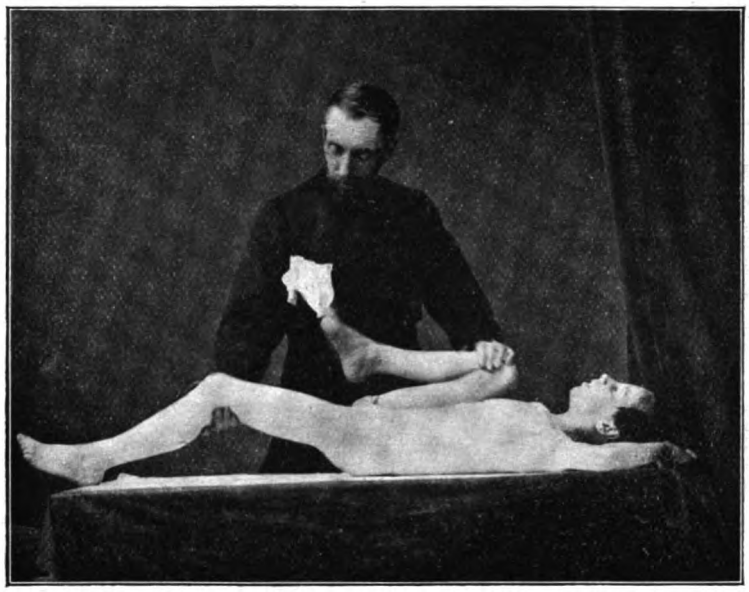
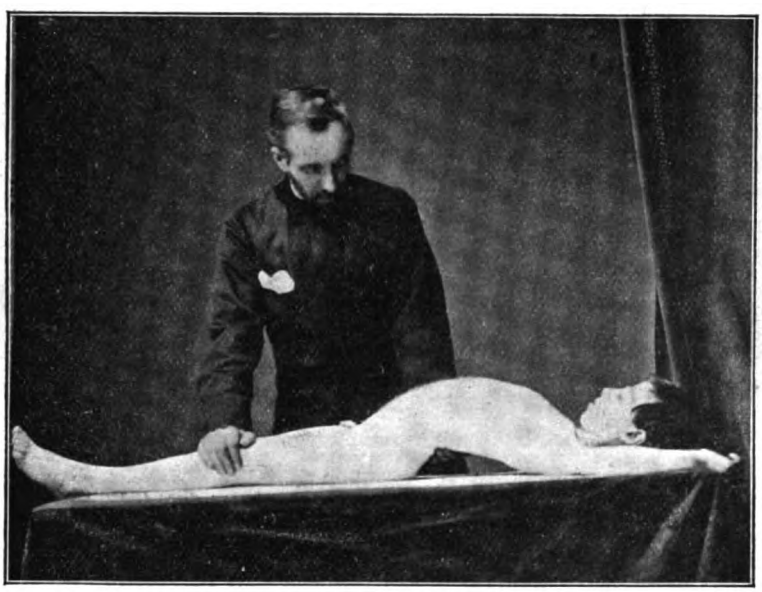


FIG. 10.



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blacksmith and a saddler, to both of whom the apparatus was unknown. At the Stanley Hospital the same process had to be gone through for a time with each splint required by me, as there was no instrument-maker in the town prepared to make them. Mr. Thomas had his splints made at home by his own blacksmith and saddler. In time, Mr. Joseph Critchley, who had learnt to make them in times of pressure for Mr. Thomas, undertook to supply them to me and to all concerned as now and heretofore. It was decided to put on a double splint, as being easier and safer under the circumstances, of the form shown you in Fig. 1. This was ready about a week later, and was applied on June 1st. The greatly flexed limb, on gentle manipulation, came fairly down into line with the trunk, the shortening being then found to be really under an inch. This is noteworthy, because I mentioned, when describing the case, that there was apparently shortening of about three or four inches, but when the shortening came to be actually tested it was found, to our surprise, to be under an inch. The hip went on very quietly and comfortably, and after three months a single hip splint for the right side was substituted. At the end of six months she was up on crutches, wearing the splint (Figs. 3 and 4), and having, in addition, a high patten attached to the opposite boot. The patten was then much more frequently used in this disease than it has been since. Mr. Thomas himself gradually found that by the time the patients could get up they were fit to bear a little concussion and the weight of the body on the diseased side; so we seldom use the patten now, except in very severe cases, or where the patients are very young children. Shortly after this, an abscess was opened over the great trochanter near the affected hip, and a sinus remained. At the end of a year some pieces of bone were pulled out of the sinus, being apparently portions of the head of the femur, but whether truly so, or derived from the great trochanter, cannot now be said. After fifteen months the sinus healed completely, and the joint got well without any stiffness. Meanwhile the left tibia and humerus gradually healed from their abscesses and small exfoliations of bone. One of the most remarkable incidents was suppuration and sinus at the ulnar side of the left wrist with issue of the cuneiform bone; this entirely healed, leaving the movements of the joint unimpaired, though with some prominence of the ulna, long before the hip got well.* In January, 1877, after treatment for about a year and eight months, this patient left the hospital and had apparently recovered; but owing to a faulty address she was lost sight of, and never seen again by me until, after a prolonged search in various parts of Cheshire, I heard of her more than twenty-five years after she first came under my notice. At an interview on May 27th, 1901, I found that she had good use of her right hip, though walking with a roll and dipping the right hip and shoulder at each step. Up to four years previously she was able to walk long distances on a level road, as many as sixteen miles on one occasion, but for two years after that time her left leg broke out again, and her walking was stopped, and when resumed had not extended beyond three miles at a time. At the interview in question this place had been healed two years. On testing the right hip-joint as she lay on her back, with the left hip and knee flexed on the abdomen (as in Figs. 8 and 9), there was found slight permanent flexion to an angle of about 120° and adduction of about 30°, but there was free movement of the joint within these limits. Her general bodily condition was fairly substantial; but she was pale, and suffered a good deal from dyspepsia, flatulence, constipation, and imperfect feeding in consequence.

The second case is that of a boy who came under my care in hospital at the age of eight, in March, 1889. Some nine months previously his right thigh and left upper arm became impaired, and were poulticed and incised at home, the patient being kept in bed during these nine months. When admitted he was suffering from suppuration and necrosis of several bones, to a small extent of both humeri, to a greater extent of the right tibia, but most of all of the right femur. With this there was stiffness of the right hip and much flexion, abduction and rotation-out of the hip-joint, great enlargement of the glands in Scarpa's triangle, and also swelling and stiffness of the right knee. The proper management of the hip-joint was now beset with difficulty, owing not so much to the progressive disease of the thigh bone, as to the faulty attitude already acquired. Thomas's single hip-splint was worn for a long time, but without much effect in reducing the limb

* Reported in the 'Brit. Med. Journ.,' October 21st, 1876.

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to line, except in the abolition of flexion. As it was, the stiff abducted hip impeded the treatment of the necrosed femur, and the necrosed femur still more prevented the due management of the stiff hip-joint. He was sent home in 1889 after three months in hospital, but returned in 1893, 1894, and 1897 for treatment of the lower limb. During each stay some operation was performed—the incision of abscess, scraping of sinuses, or removal of necrosed bone. On one of these occasions, in November, 1893, while excavating the femur I wounded the femoral artery, lying in densely matted tissues that made ligature there impossible. The wound was plugged, and the patient nearly died from collapse, but recovered. He was again in hospital under my care as recently as November, 1901, with a periosteal abscess of the right scapula. This was incised and found to have bared the bone in the infra-spinous fossa and spine itself. After dressing with a packing of cyanide gauze, it healed in a few weeks. He is now an active young man of over 21, and although the abducted right hip has been followed by a curve in the femur during the healing of the necrosed shaft, the latter deformity serves as compensation to the former in bringing the limb alongside the other. I now throw on the screen a photograph (Fig. 11) of the patient taken early in February, 1902.

He has been placed so as to show the pelvis symmetrically as nearly as possible. You will notice that the left lower limb is a very strong and well-developed one. The abducted right hip-joint is, I find, firmly ankylosed, and during recovery from the necrosis of the shaft the weakened bone has undergone a compensatory adduction which is now practically of great use to him. You see there is also shortening of the left humerus owing to necrosis and arrested growth there, and you can, I think, just see a scar over the right humerus, owing to a very slight disease there. The next photograph (Fig. 12) shows the same patient from behind. You can see the numerous scars behind the right hip and over the thigh. There is a scar behind the thigh near the left sacro-iliac joint, but that seems to have run its course without much notice. You can also see the spot on the right scapula, where the abscess of last November was found, which had recently formed; it is now totally healed. I have not the slightest doubt that this was a case of multiple suppurative osteomyelitis and periostitis from the first. Of course, if one could have put the patient from the beginning into an effective splint, quite straight, it would not have mattered whether ankylosis occurred or not.

The third case is that of a boy æt. 11, who was admitted into my ward at the Liverpool Royal Infirmary on February 11th, 1898, on account of a large acute abscess of the left thigh and hip region attributed to a blow received a fortnight before. There was flexion of the left hip-joint, and accordingly disease of the joint established at the time, but its pathological origin was not made out, and remained obscure for some months. Aspiration was performed three times during the first week, and twice subsequently at intervals of a week. The amount of pus withdrawn diminished from nine and a half ounces to two fluid ounces, resulting in almost complete drying up of the large abscess. But the aspiration was attended on the first occasion by a great swelling of the whole limb, apparently due to acute phlebitis, which I could only attribute to inoculation with pus or other material in the act of puncture. This slowly subsided on the left side, but at the end of three weeks œdema appeared in the opposite leg, followed by a general swelling, especially of the thigh. This appeared to me to indicate further phlebitis and thrombosis, which I concluded had spread up the left common iliac vein and down the right. Some ten weeks after admission there was evidence of abscess in the right thigh, and on aspiration fœtid pus issued. Under chloroform this was incised deeply on the outer side, but found to be nearest the surface on the inner side of the limb over the vessels where a counter opening was made. The bone was free from disease, so the abscess was concluded to be that of a peri-phlebitis. The large wounds now left were packed with cyanide gauze during many weeks. The condition of the patient became very grave owing to fever and emaciation. The labour involved in the frequent necessary dressings was also great, and my story would be very incomplete were I to omit a proper tribute to the continuous and patient care of Miss Handley, the sister of the ward. He passed into a condition from which there appeared little or no hope of recovery. Meanwhile the left hip-joint, noticed on admission, had now become evidently in need of mechanical support. I wished to put on a Thomas's splint,

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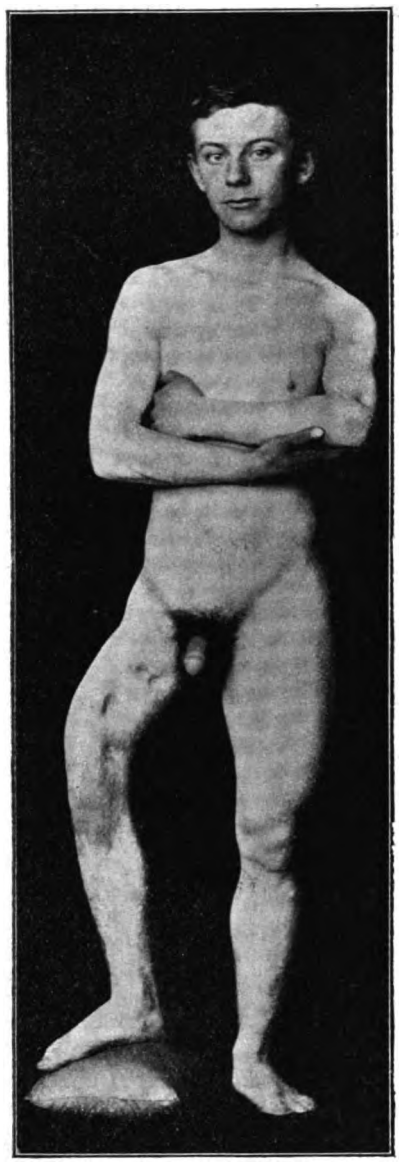
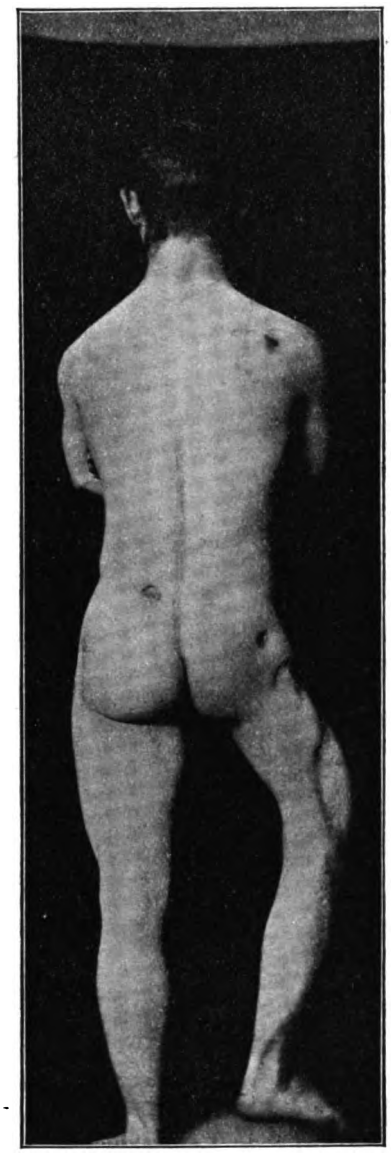
and foresaw the pathological adduction, which is one of the great drawbacks in the treatment of hip-joint disease; for even if the treatment be successful, there is sometimes a tendency to adduction of the affected thigh. But the delicate state of the patient, the formation of abscesses, and the

FIG. 11.



numerous dressings required, showed that the presence of a splint would be, if possible, a greater evil than its absence. The ward sister having had impressed upon her my anxiety to treat the limb in the abducted position, took advantage of the helpless immobility of the patient to further fix him with sand-bags so as to keep the limb abducted as much as possible. She succeeded in keeping him alive and in certainly preserving for the limb a very good position under the circumstances. The boy emerged from his

FIG 12.



danger, fattened, and eventually improved so greatly in health that I felt able to undertake seriously the mechanical management of the hip, which had lapsed somewhat into adduction as the boy became able to move himself. Accordingly in

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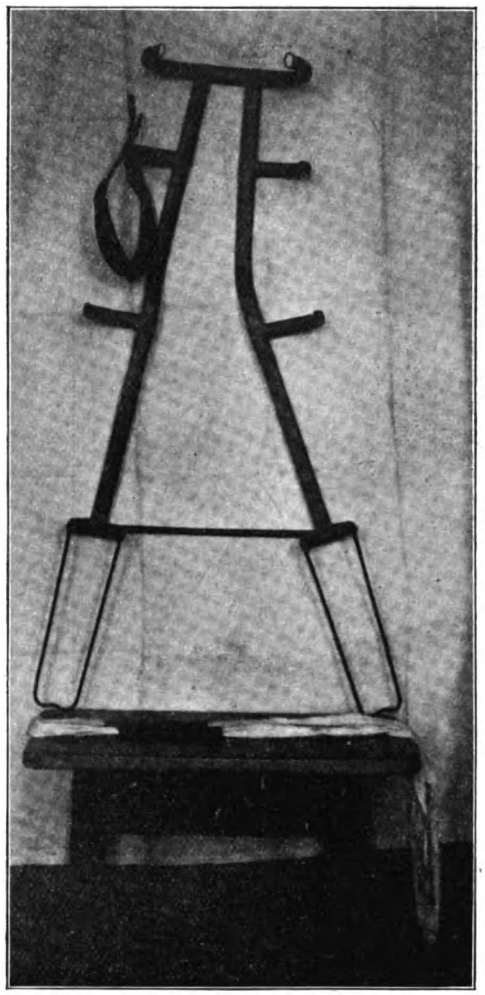
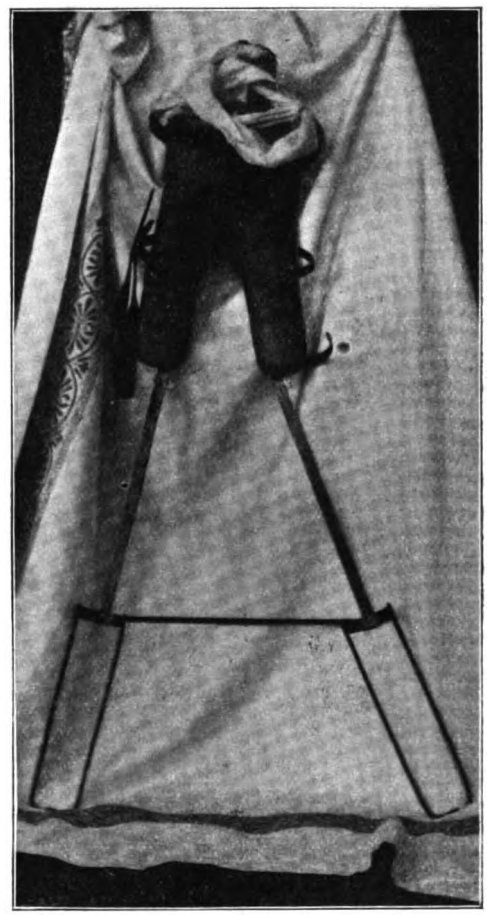
November, 1898, more than nine months after admission, I had prepared for him a suitable splint to fix the trunk and both lower limbs in the position required. The splint is a sort of double Thomas's hip and knee splint combined, as suggested by Mr. Robert Jones for the express purpose of correcting abduction deformity in hip cases, and

FIG. 13.



I cannot speak too highly of it. I now show you (Fig. 13) a sort of double hip splint, but its lower ends are continued in the form of a portion of Thomas's knee splint. Each longitudinal portion is applied to one lower extremity, and the lower ends are kept very considerably apart by means of an iron rod which fixes them firmly in the position required. On the side required, the apparatus has an extra abduction attitude imparted to it. Attached to each longitudinal bar there are certain peculiar side pieces which clip different portions of the body. One is the usual crescent-shaped piece which goes round the trunk at the site of the angle of the scapula, and they are all fairly flexible flat iron rods which grip the trunk and help to keep the splint in place. Attached to the upper side piece on the right in the photograph is a

FIG. 14.



leather perineal band. This goes under the perineum of the sound side, because it is not convenient to attach the perineal band on the diseased side, especially if the operation of osteotomy has to be done, as it was in this case.

I now show another form of the same splint (Fig. 14), some portions of which are not permanently padded, but there is a comfortable, loose pad interposed for the back and upper part of each

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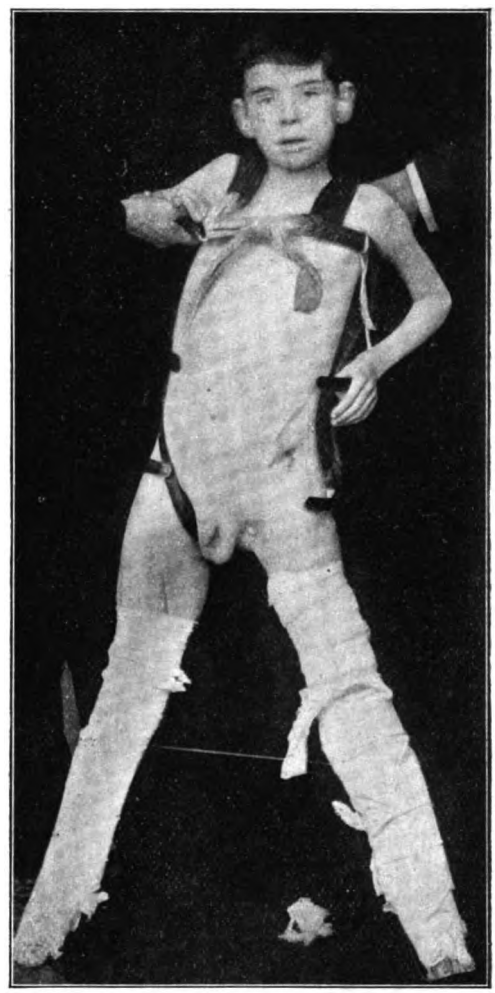
thigh. The padding is of cotton wool and is enclosed in basil leather.

The next photograph (Fig. 15) represents the patient taken while wearing the splint. The portion lying behind the affected limb is specially abducted to an extra degree. The patient was put under the influence of an anæsthetic and the abduction of the deformed hip forcibly attempted. This I could not do in the present case, so I proceeded at once to osteotomy of the femur as near the hip joint as I could get, the chisel being driven in above the great trochanter several times through the same aperture in the skin until the neck of the bone seemed to divide. The whole operation was performed under antiseptic precautions, the small wound sewn, a cyanide gauze dressing put on, after which forcible abduction was repeated, and the limbs fixed to their respective arms of the splint. Healing was by first intention. Each limb was separately enveloped in rolls of cotton wadding bandaged on, and then applied to the splint. The limb under special treatment was additionally provided with extension bandages fixed on by strapping and attaching to the lower end of the splint, which reached a good distance below the foot. Counter-extension was kept up by the perineal band of padded leather fixed to the opposite side of the body so as to be out of the way of the seat of operation and dressing. A well-constructed pad, as shown in Fig. 14, was interposed between the back and buttocks, so that the splint itself did not require any fixed padding. The patient was then fixed firmly to the splint, with legs astride as if attached to a St. Andrew's cross. If at any time tired of lying on his back he was turned over on his face, with the splint uppermost for a change. On a subsequent occasion, having to explore a sinus over the hip bone, I removed some fragments of bone, and then for the first time began to suspect the origin of the disease. The articular deformity and the progress of the joint conformed in several ways with the course of a tubercular case, but the acute abscess at first and the rapid onset of the whole thing were inconsistent with tubercle. Acute suppurative osteomyelitis appears to me to account for everything, and I have no hesitation now in assigning this as the pathology of the case. After about two years in the Royal Infirmary at Liverpool he was transferred for about two more to a Children's Convalescent Home at West Kirby, Cheshire, suffering from severe albuminuria, anasarca, anæmia, and purulent discharge from several old sinuses in the left thigh. The limb was in good position on a splint, and the nurses kept the sinuses carefully cleaned and frequently dressed. Dr. Charles Macalister, of Liverpool, had charge of him for medical purposes, and succeeded admirably in gradually improving the state of his health,

Fig. 15.



reducing the albuminuria by a judiciously spare diet, chiefly of milk, and by other means with drugs. Mr. Robert Jones had charge of the surgical aspects of his case, and eventually dispensed with the splint and allowed the boy up. From time to time I visited the boy, and witnessed the progress he made under the careful treatment of my two friends. The boy is now fairly well-

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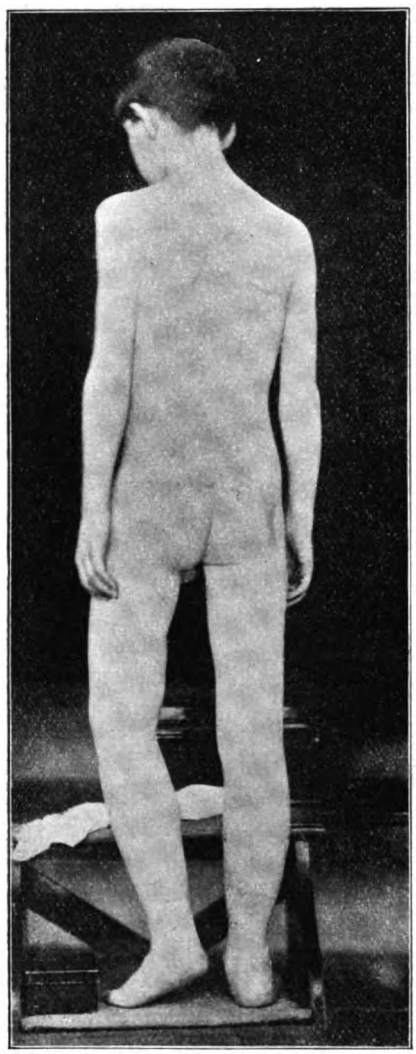
grown and vigorous, with only a trifling discharge from a sinus. I have passed round photographs taken early in February, 1902, in two attitudes. I have also a further small point to bring to your notice in those photographs. You will perceive in each flank a swelling that is evidently caused by dilated veins, showing how part of the collateral circulation was carried round.

FIG. 16.



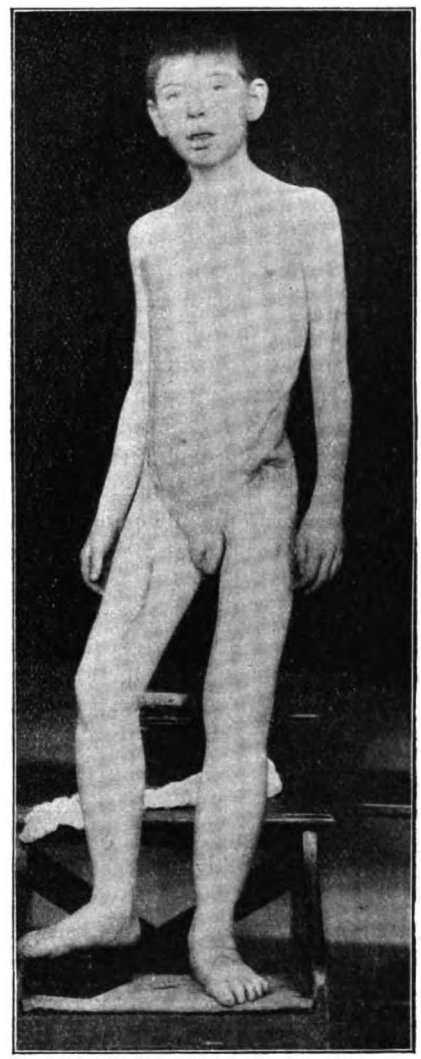
I have now merely to suggest the following conclusions:—

Cases of hip disease, from whatever pathological cause, should receive mechanical treatment in the straight position; the earlier the better, and for as long as required. In many slight cases nothing more is needed.

In case No. 1, though unusually deformed at first, recovery in fair position in spite of bone suppuration occurred in consequence of the splint, and could not possibly, I think, have occurred without a splint having a similar effect.

In case No. 2, for want of early mechanical treatment, permanent deformity, fixed by ankylosis, occurred.

FIG. 17.



Tubercular abscesses do not contra-indicate Thomas's splint, but in case No. 3, mechanical treatment at the early period when most required was made impracticable owing to an accumulation of difficulties, but was successfully adopted later, and adduction deformity corrected by osteotomy and forcible abduction.

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CANCER: ITS ORIGIN, NATURE, AND GENERAL PRINCIPLES OF TREATMENT.

(*Debate continued from page* 128.)

THE following communication was then read to the meeting by the senior Hon. Secretary, Dr. A. Cooper.

Contribution to a Discussion upon the Parasitology of Malignant Growths (on Syncytioma malignum).

By J. GEORGE ADAMI, M.D., McGill University, Montreal.

MR. PRESIDENT AND GENTLEMEN,—Asked most courteously by your committee, in default of being present, to contribute something to this evening's debate, and recognising the difficulties under which both my audience and myself must labour when I accept, it has seemed to me better not to attempt an academic essay covering the whole territory of the possible or presumed relationship of parasites to malignant growths, but to consider more in detail one small portion of the vast field before us, in the hope that this will not already have been traversed by other participators in the debate: in the hope further that the deductions which may logically be drawn from the study of a single form of tumour may throw light upon the essential nature of tumours in general. And in advance let me say that I am well acquainted with the ancient Greek parable of the certain man who, having a house to sell, circumambulated the city, bearing in his hand a brick as sample. I trust that when I have said my few words the brick will not be thrown in my face. Until recently we have been in the dark as to the relationship between the embryo and the maternal tissues in the womb; now certain important points are well-established. The observations of H. Peters, of Vienna, upon the earliest human ovum which has thus far been discovered in utero—an ovum of the third or fourth day—have proved in a manner admitting of no dispute (when compared with what we know concerning mammalian ova in general) that even at this early date the cells of the ovum have a marked influence upon the uterine mucosa at the spot where the ovum becomes arrested. The epithelial cells of that mucosa are so acted upon that they become dissolved and disappear, and the ovum from being on the surface sinks down into a depression, becomes imbedded in the uterine wall, so that now it lies in close apposition to the capillaries underlying the epithelium. These observations have been largely confirmed by my late colleague, Dr. J. C. Webster, now of the University of Chicago.

This is but the first step in the formation of the placenta. The outer layer or layers of the epiblast of the rapidly growing embryo, termed by Hubrecht the Trophoblast, continues this process of erosion and infiltration in a most remarkable way. This trophoblast literally eats into the maternal tissue opposed to it, forming at first solid cellular processes, purely epiblastic; later, as the chorionic mesoblast and the allantois with its vessels become developed, these processes gain a mesoblastic and vascular core; so are evolved the characteristic chorionic villi. Both the more primitive solid processes and the villi, through this phagocytic, or more truly extra-cellular digestive action, of the trophoblast, make their way into the maternal uterine veins and venous sinuses, whereby the cells of the embryo come to lie within the maternal blood-spaces and gain their nourishment, and nourishment for the embryo in general, directly from the maternal blood.

There was for some years violent debate, more especially in Germany, as to the nature of the trophoblast, whether it was of fœtal or maternal origin. This culminated in an important debate between anatomists, zoologists, gynæcologists, and obstetricians at the 1897 meeting of the German Naturforscherversammlung. The most that the most violent opponents of the fœtal view can now bring forward is that not all the cells considered by some as trophoblastic or syncytial are of fœtal origin. Some, they say, are maternal. Nay, more, in the course of a most painstaking and minute study of the development of the rabbit's placenta, my colleague, Dr. Chipman,* has shown in a conclusive manner that these trophoblastic or syncytial cells,

* Through the liberality of the Governors of the hospital, Dr. Chipman's Edinburgh thesis, with its abundant photographic illustrations of this and kindred points, will be published in the series of 'Studies from the Royal Victoria Hospital.'

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when they gain entrance into the maternal blood sinuses, passing along the walls digest and replace the endothelium of those sinuses, so that eventually there is developed a most complicated system of passages lined by cells of fœtal origin, and containing maternal blood, or almost, in the words of Matthias Duval, the placenta is to be regarded as "a maternal hæmorrhage encysted by fœtal tissue." By this remarkable "adaptation" the fœtus gains nourishment from the maternal blood.

I have described this at some little detail because I wish to impress upon you that here, as a normal or physiological condition, we have passing those identical processes of invasion and infiltration which we have been accustomed to regard as peculiar to, nay, as the essential feature of malignant growth. The only difference between what is here seen and malignancy is one of degree, or perhaps, more correctly, of orderliness. This infiltration is due to the inherent properties of the syncytial cells. Nor would this appear to be by any means the only case of antagonistic "phagocytic" cellular activity. Metchnikoff has of late been engaged in collecting evidence to prove that such activity is fairly general within the organism, and is the basis of senile changes.

Under ordinary conditions this syncytial invasion is strictly limited; it only affects a definite area of the uterine wall, and with parturition and the separation of the placenta the whole area of invasion is separated off and discharged. In other words, to explain these normal phenomena (if the phrase be permissible), we have to recognise the existence and interaction of two forces—on the part of the fœtal cells of "invasive" properties, on the part of the maternal tissues of protective properties.

Having laid this down, I may now approach my main point:—Occasionally—and apparently not so very occasionally, for Marchand collected from the literature some sixty-five cases, recorded between the beginning of 1895 and the middle of 1898—these syncytial cells, after normal labour, or more frequently after premature labour, are found to take on aberrant growth, to trespass beyond the placental site, to infiltrate extensively the uterine walls, the vaginal tissues, the pelvic tissues in general, and, what is more, to form secondary growths in distant organs, the lungs, the spleen, the brain, and so on. These syncytial cells are large and characteristic; there is nothing quite like them. We have, that is to say, as nearly perfect histological evidence as it is possible to possess, that at least one group of primary malignant tumours of the uterus—a group, I may add, presenting "exquisite" or "eminent" malignant properties—consists of the aberrant growth of fœtal cells within the maternal organism.

This view of the fœtal nature of the cells of the deciduoma malignum was, I need scarce say, violently opposed for many years by an important section of pathologists; but now Marchand, whose opinion cannot be passed over, has been forced to accept the view: in short, no other view is possible at the present time. The typical deciduoma is a syncytioma or chorio-epithelioma malignum. I greatly doubt whether any Committee of the London Obstetrical Society could be found in these latter days to report, as one reported only five years ago, that a group of specimens of this form of tumour was of connective tissue origin, and so sarcomatous.

Can we explain this most remarkable form of neoplasm by any parasitic theory?

To do this it is obvious that we must start from the assumption that micro-parasites, in some way or other, exalt the already existing invasive properties of the syncytial cells, so that now they invade and infiltrate to an excessive extent. That must be the starting point.

For myself I am perfectly prepared to accept this much, namely, that parasites and their products are capable of stimulating cell growth. I see that in his recent Milroy Lectures Mr. C. Powell White discovers a poorly defended spot in my argument published last year, in that there I make this same admission, while simultaneously I lay down that the vegetative and proliferative activities of the cell are inherent, the functional activities set up by stimulation from without. Upon first consideration the objection appears valid. Further consideration, I think, shows that it does not hold. We can imagine stimuli leading to building-up—anabolic—processes in the cell, processes which, under ordinary conditions, are preparatory to functional and katabolic processes, which in the absence of circumstances favouring katabolism, favour cell proliferation. Now, in connection with this very form of tumour we are considering, it is not a little interesting to note that the growth does not occur in the placenta during the period of pregnancy, during the period,

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that is, when the syncytial cells are actively performing their functions of absorption and excretion, but afterwards, when these same cells, not having been shed, are left in the condition of gaining nourishment—of being stimulated from without also—and of having no opportunity to perform their normal functions. Under such conditions microbic products *might* favour overgrowth. It will be seen that the two statements are not contradictory.

But is it necessary to call in micro-parasites to explain the aberrant growth? There, I confess, looking at the matter in the abstract, I feel more than doubtful. For, in the first place, it will be seen that this assumption—and remember that it is still only an assumption, and that, consequently, one is permitted to discuss it in the abstract—this assumption demands that microbes of one or other order have the peculiar power of so acting upon the cells of one organism—the embryo or fœtus—that they invade the tissues of another organism—the mother. Our knowledge of the phenomena of transplantation and implantation shows that it is not necessary to call in such external agencies. That micro-parasites should have this power is, to say the least, unexpected. At most we must admit that it is remotely possible.

In the second place, one of the favourite arguments of those who hold parasitic theories is the similarity or analogy between the local growth and metastases of malignant neoplasms and the primary focus of infection, its local extension and metastatic granulomatous, or abscess developments in the case of bacterial infection. Undoubtedly the parallelism is close, so close that we may admit parasitism as the active agent in both conditions. Indeed, these syncytiomas demonstrate most convincingly why this is so. Bacteria are, we know, the causative parasites in the one: what these syncytiomas show, and show without possibility of denial, is that the trophoblastic cells—the tumour cells—are the parasites in the other. They are cells of foreign origin; it is they that invade; they that gain entrance into the blood stream, that get carried to remoter parts of the organism, and there set up degenerative and reactive processes, and so bring about a condition of affairs resembling what is seen when bacteria are the invading agents. *The tumour cells are the parasites;* and if we admit this—and we must admit it—then the analogy becomes absolutely valueless as an argument in favour of the micro-parasitic origin of malignant neoplasms. To urge, that in order to induce the aberrant growth, there must be parasites within the parasites, is to pile Ossa on Pelion—it is supererogatory.*

Nevertheless, these considerations, while demolishing the argument based upon analogy, do not wholly do away with the possibility that micro-parasites play some part in the production of these and other malignant growths. There is yet another method of approaching the subject.

As I have already laid down in discussing the normal growth of the syncytium, we have to see that there are two factors constantly opposing each other—the invasive powers of the syncytial cells and the self-protective powers of the maternal tissues. This is true, also, of the abnormal growth of the same. It is possible, therefore, that the aberrant growth of the syncytioma may depend not so much upon an exaltation of the invasive powers of the component cells as upon a lowering of the self-protective powers of the surrounding maternal tissues. Indeed, the observations of Beatson and others, even if therapeutically they have not been crowned with complete success, can only be regarded as demonstrating that this second factor has to be taken into account. Alteration of general metabolism following upon removal of the ovaries does at times arrest malignant tumour growth. Indeed, at the present time I feel even more strongly than I expressed myself a year ago, that it is along the lines of research into the exaltation of the protective mechanism of the organism, and into the diminution of the proliferative powers of the tumour cells, that our energies should now be directed—and coincidently I note that the last twelve months have not yielded any more promise than showed itself then of distinct help to be gained from this weary and unsatisfied hunt after specific cancer parasites.

I do not take the stand of denying the existence of such. In this special connection we can observe a parallelism between the virulence of the microbe in bacterial disease and the protective powers of the organism on the one hand, the

* It is worthy of note, further, that in the known cases where we have intracellular parasitism—*e.g.* coccidiosis,—we have no sign of the cells themselves taking on these parasitic functions.

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invasive powers of the cancer cells and the protective powers of the organism on the other. But have we any sound grounds for assuming that the presumed cancer parasite acts by stimulating and augmenting the invasive powers of the cells, or by doing the reverse, namely, by lowering the vitality of the tissue about to be infiltrated?

Looked at thus closely it will be seen that this conception of parasite acting within parasite leads to marvellous complications. I may be wrong, but I think that I have noted time and again that when we determine fundamental laws and fundamental phenomena they are wonderful in their simplicity, so that now when I find that a conception followed up leads us deeper and deeper into the mire, I shrewdly suspect that the conception is wrong. So it is with the parasitic theory of malignancy as now held by so many. It may be right, someone may discover the germ or germs—but neither on theoretical grounds nor on the results of practical observations can I base myself with any satisfaction. Certainly Gaylord's results upon this side of the Atlantic do not carry conviction, and neither he nor Plimmer, so it seems to me, have proved that their bodies are any other than degenerative products, the abundant presence of numerous forms of which in tumours was first demonstrated by Fabre-Domergue, and later and most fully by Pianese.*

My inclinations at present, it will thus be seen, in the absence of more definite discoveries, lead me to doubt very gravely the permanent presence of micro-parasites in malignant growths; nor do I think that a study of the deciduoma malignum can lead to any other than a firm belief that alterations in cell properties and assumption of the parasitic habit by cells is, I will not say, at the bottom of malignancy—for we have still to determine what initiates the alteration in property—but must be recognised and must be taken as the basis for future advance.

Thus to sum up what, I think, may be legitimately deduced from a study of the syncytioma malignum:

1. There exists one form of tumour of highly malignant type in which the infiltrating cells are not those of the organism itself, but are derived from another organism.

2. The infiltrative and invasive properties of these cells are not a new acquirement, but are an exaltation of properties normally possessed by them; or, more exactly, under normal conditions we observe that there is an interaction of two forces: the invasive properties of these cells, the protective properties of the surrounding maternal tissues, by which interaction the extent of invasion of the cells is strictly limited to the placental site.

3. The development of the syncytioma malignum must therefore be attributed to either an increase in the invasive properties of the syncytial cells, or a lessened resistance on the part of the maternal tissues, or a combination of the two.

4. If micro-parasites play any part in the production of the tumour, that must be either by exalting the infiltrative powers of the one or by lowering the resistant powers of the other.

5. Proof positive of the existence of such specific micro-parasites is still wanting, and in its absence it is difficult to conceive how specific micro-parasites should bring about these results.

6. Admitting that micro-parasites and their products are capable of inducing cell proliferation, we must also admit that other external agencies have the same capacity, so that micro-parasites, at most, may be one of the causes of aberrant cell-growth.

7. The prevalent conception of cancer parasites, as existing within the cancer cells, involves the idea of malignant growths being the product of parasites acting within parasites; for, as shown by this study of the syncytioma, the tumour cells themselves are essentially parasitic in the organism.

In short, tracing the present popular idea to its legitimate conclusion, the most that can be said for it is, as the association of parasitic ideas may already have reminded some of my audience, that it is in harmony with the great generalisation of the poet, that—

Great fleas have little fleas upon their backs to bite 'em,
And little fleas have lesser fleas, and so *ad infinitum*.

Is this generalisation also to be applied to explain the action of pathogenetic bacteria? I am willing to see, in the long-continued action of micro-parasites, a process which may, like other modes of stimulation, initiate aberrant and neoplastic cell growth, but beyond this point we seem to be asked to contemplate a most extraordinary state of affairs.

* Such bodies, described as Blastomycetes, have, I should add, been figured by Rossi-Doria as found by him in two cases of deciduoma malignum.

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THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

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* *Specially reported for The Clinical Journal. Revised by the Author.*

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A CLINICAL LECTURE

ON

ACUTE PERITONITIS.

Delivered at Guy's Hospital, May 21st, 1902.

By W. HALE WHITE, M.D., F.R.C.P.

GENTLEMEN,—I have taken the subject of acute peritonitis for to-day, because from the very nature of the cases so many of you hardly ever see one. The patients are so urgently ill, and operation is nowadays almost the only thing which can save many of them, so that by the time you come to the ward you find the patient has already been operated upon. Probably most of the cases of acute peritonitis which come in here are operated upon within a few hours of their admission, so that all that you see is a patient with a number of bandages around him or her. The reason why the patient is so severely ill is not difficult for you to understand, because if you will think for a moment you will remember that the peritoneum has an enormous area. The area of the peritoneum is about equal to that of the skin, so that you see there is a very large absorptive surface, and effusion and absorption are probably constantly going on in the peritoneum, for, as you know, it is always just slightly moist, so that the friction of the various parts one upon another is rendered very easy. A good deal of work has been done lately on the question of effusion into and absorption from the peritoneal cavity, and many people, I think, have the misconception that the chief determining factor—and they often think the only determining factor—of effusion into the peritoneal cavity is an alteration in the blood pressure of the portal area. But if you think for a moment, you will understand that that is not so; for were it so a person with congenital heart disease, and therefore with high blood-pressure in the venous

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system, should have effusion into the peritoneal cavity. Further, it can be demonstrated experimentally that it is not so; because if you ligature the portal vein you do not get effusion into the peritoneal cavity. But supposing you take a dog in whom you have ligatured the portal vein, and you dip a little piece of intestine into ordinary tap-water, you will find there is effusion into the peritoneal cavity. That experiment, therefore, teaches that while increase of venous pressure will not in itself cause effusion, it is a contributory factor, because the venous pressure being increased, the slight damage to the peritoneum which follows the application of tap-water leads to effusion into the peritoneal cavity. The experiment also teaches you that for effusion to take place into the peritoneal cavity the permeability of the peritoneum itself has in some way to be altered; and even the slight alteration which ensues upon dipping it into tap-water is sufficient to determine effusion into the cavity.

Then, with regard to absorption. Much work has been done of late years with regard to absorption from the peritoneal cavity, and this absorption is undoubtedly very rapid. A controversy has raged around the point whether absorption takes place chiefly by the blood-vessels or by the lymphatics, and that question cannot be said to be settled entirely. Experimenters, introducing large quantities of fluid into the peritoneal cavity, have claimed that absorption takes place only by blood-vessels. But since then other experimenters, introducing lesser quantities, have claimed that if you introduce only a small amount, absorption takes place by means of the lymphatics. The introduction of a smaller amount of material is much more comparable to what is constantly happening in peritonitis than the sudden injection of many ounces of fluid into the peritoneal cavity. There are at least three factors concerned in absorption from the peritoneal cavity, all of them of importance in connection with peritonitis. And, first, I would mention what we know little about, namely, absorption by means of stomata between the endothelial cells lining the peritoneal cavity. Another factor is filtration, and that will depend on the pressure within the peritoneal cavity. Hence it is important to remember, in connection with peritonitis, that intra-abdominal pressure is greatly increased by the movements of respiration. They are very much lowered in peritonitis, and so absorption is retarded; on the other hand, it will be increased by the distension of the intestines with gas, which often takes place in peritonitis. And the third factor which you have to do with is osmosis, which varies inversely with the alterations in the specific gravity of the fluid which is about to be absorbed. Of course, only hypotonic fluids can be absorbed by osmosis, and, generally speaking, the rate of absorption of a hypotonic fluid increases in the inverse ratio of its concentration. We cannot here discuss further the many difficulties connected with thoroughly understanding the question of peritoneal absorption. They merge into the discussion of the differences between vital and physical processes.

With regard to two little points in connection with man. It appears that, at any rate in man, much absorption takes place by means of the lymphatics of the diaphragm, and the glands in the anterior mediastinum are secondarily affected in acute peritonitis, absorption taking place through the lymphatics of the diaphragm, and being passed on into the lymphatic glands of the anterior mediastinum. And the other factor which you are likely to forget in thinking about acute peritonitis is the great importance of the omentum. When he was Gull research student, H. E. Durham made many investigations, and showed that in cases of acute peritonitis the process often first attacks the great omentum, and post mortem the greater part of the disease may be found to be connected with the omentum, and not with the rest of the peritoneum. So you have not made a post-mortem on a case of acute peritonitis properly unless you have carefully investigated the omentum. Also it has been shown recently that the omentum is of great value in another direction, namely, it is an organ which is used by Nature largely to attempt to heal in cases of peritonitis, for if there has been a rupture of any viscus it is not unusual at the post-mortem to find that the great omentum has applied itself to the rupture, and has apparently attempted to seal it.

The next thing necessary to consider when you are thinking about acute peritonitis is the following. You cannot form a proper conception of a case of acute peritonitis unless you have some clear notion in your mind as to its etiology. When I was a student it was a much disputed point whether there was such a disease as idiopathic peritonitis.

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Now it is generally admitted there is no such disease as idiopathic peritonitis; but only quite recently I saw in one of the journals an attempt to resuscitate idiopathic peritonitis, the author suggesting that many of the cases of so-called rheumatic peritonitis were idiopathic. But unfortunately for that notion, as clinical observers we know nothing of the so-called rheumatic peritonitis; it is always a diagnosis covering ignorance, there is no evidence of it whatever, and I have been to the trouble to look up the accounts of many of the older cases of so-called rheumatic peritonitis, and none of them which I have come upon will bear modern-day criticism. For instance, there was a case recorded by Dr. Fuller, who gave an account of what he called rheumatic peritonitis; but as the patient had had pneumonia also there is little doubt that nowadays that would be regarded as a case of pneumococcal peritonitis. So I must ask you to dismiss from your minds any such diseases as idiopathic peritonitis, or rheumatic peritonitis.

I have written out a list of various micro-organisms which are the cause of peritonitis. I will write them on the board. As you know, the most common micro-organism as a cause of acute peritonitis is the bacillus coli communis. That is very frequently found. There is also the streptococcus pyogenes, which is almost as common as the bacillus coli communis. Then, but considerably less frequently, there are staphylococci, pneumococci, and gonococci. Much rarer organisms which may be found in the condition are the micrococcus lanceolatus, the bacillus proteus, bacillus aërogenes capsulatus, bacillus pyocyaneus, and bacillus typhosus. It is important to remember that a pure cultivation of bacilli from the peritoneum is rare; most cases of acute peritonitis are mixed infections. It does not matter at all how these micro-organisms are introduced into the peritoneal cavity. The simplest mode of production in man would be a stab with a dirty dagger, and occasionally acute peritonitis is produced in that way. But that is of course quite the exception, at any rate in this civilised country. The way in which acute peritonitis usually arises in England is by perforation of some hollow viscus within the abdomen. In order to show you the importance of that, I may mention that I came across a collection by Jantz, of twenty-one cases of perforation-peritonitis, and it will be a matter of great clinical interest for you to see how they group themselves. Out of the twenty-one, seven followed perforation of the appendix; five followed perforation of a gastric ulcer; three followed perforation of a typhoid ulcer; two followed hernia; one followed rupture of the stomach; one followed rupture of the intestine; one from rupture of a duodenal ulcer; and one from rupture of a perimetritic abscess. That, I think, represents with sufficient accuracy the relative frequency of the causes of ordinary clinical perforative peritonitis. Certainly it corresponds with my own clinical experience, for in that the commonest cause is perforation of the appendix; the next commonest is perforation of gastric ulcer; the next, perforation of typhoid ulcer; and then there are the rarer causes. But all sorts of rare causes may lead to the condition. For instance, I have seen perforative peritonitis secondary to a suppurative lymphatic gland, which gland was suppurating because of an inflamed pile.

The next most frequent cause of peritonitis is infection through the female genital organs. You know that is very likely to occur, because of the Fallopian tubes opening out as they do. Some years ago that form of this variety of peritonitis, which is called puerperal peritonitis, would have been regarded as more frequent than perforation peritonitis. But that was in the days before it was learnt how important it was to have cleanly methods of midwifery. With that recognition there has been a great improvement in the figures of puerperal peritonitis. But I will give you some figures for the years 1875 to 1885 from the Charité Hospital in Berlin. There the number of cases of puerperal peritonitis was 176, as against 152 of perforative peritonitis. The micro-organisms most frequently found in puerperal peritonitis are streptococci, as you would expect, and the bacilli coli communis is also present. In some cases gonococci are found also. Puerperal peritonitis is not the only form of peritonitis which you would be likely to get from infection by means of the female genital organs. Occasionally gonorrhœal peritonitis occurs. Usually that is not a very severe form, and it has been said that it may be a pure infection. Some observers claim to have produced pure gonorrhœal peritonitis in animals; but it is probable that in human beings gonorrhœal peritonitis is usually a mixed infection. Although rare, it is a

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condition which you have to be on the look-out for in women.

But even this does not complete all the forms of peritonitis which we meet with clinically; experimental research has shown, as you would expect, that bacteria may sometimes pass through the wall of the intestine, even when no perforation has occurred. Clinically, in medicine we recognise that as happening in two common diseases. There may be an acute general peritonitis following upon disease of the appendix without any actual perforation of the appendix. In such a case the bacilli coli communis may pass through the damaged wall of the appendix and set up a general acute peritonitis; and you certainly can get, occasionally, a peritonitis following upon typhoid without any actual perforation. In such a case the micro-organisms pass through the thin injured wall of the gut and set up peritonitis. Only quite recently I came across a collection which had been made of such cases. Then, too, there are the rare cases in which actinomycosis affects the peritoneum. That is another instance in which the micro-organism passes through the wall of the gut without forming any actual hole—at all events, one which is to be seen by the naked eye—and peritonitis follows.

Now, you see all these forms of peritonitis—traumatic, perforative, through the female genitals, and those in which micro-organisms pass through the wall without there being a hole in it, are all forms of peritonitis produced locally, and acute peritonitis usually occurs without there being any disease outside the abdominal cavity. But you may have micro-organisms, part of a general infection, conveyed by the blood to the peritoneum, and they may pass through the vessels into the peritoneal cavity, and thus set up general peritonitis. Instances of that are pneumococcal peritonitis, about which there has been much written of late years. I find that Netter, in 140 observations on peritonitis, found it to be pneumococcal twice; this form may be secondary to pneumonia; or, in still more rare cases, of which we have had one here in the last few years, it may be a pneumococcal peritonitis without any pneumococcal infection of any other part of the body.

But even this does not finish the etiology, because you have much to think of besides your micro-organisms when you are thinking of acute peritonitis. There is no doubt that many and many a time micro-organisms get into the peritoneal cavity without any peritonitis following. They probably do so in many an operation, however careful the surgeon is, but the micro-organisms die within the abdominal cavity if there is no suitable medium for them to flourish in. Hence, if the absorptive power of the peritoneum is very good and quick, it is possible that any fluid which may be there and might form a growing medium for organisms, is absorbed, so that the micro-organisms die because they have nothing to grow in. Again, you have also to remember that the resisting power of the peritoneum varies very much. There is on record a case of a mass of fæces in the peritoneal cavity getting completely encysted; such was the resisting power of the peritoneum in that case that only a local peritonitis followed. On the other hand, sometimes the resisting power of the peritoneum is very low. You see that strikingly shown in the acute peritonitis which is one of the recognised terminations of Bright's disease. Again, you see it when not only the resisting power of the individual is lowered, but when the local resisting power of the peritoneum is lowered. For instance, it is not uncommon for the terminal event in tubercular peritonitis to be the onset of acute peritonitis. That is an instance of the local resisting power of the peritoneum being lowered.

Another point you have to think of in connection with acute peritonitis has been much discussed by the experimentalists, and that is, whether chemical irritants can set up an aseptic peritonitis. That is interesting to laboratory workers, but it has very little clinical interest. In the laboratory it is said to be possible, but it hardly ever occurs clinically. There are one or two cases on record, for instance, in which it has been claimed that aseptic peritonitis has followed from the irritation produced by the bile in a case of rupture of the gall-bladder from traumatism; but they do not count for much, I think, because we know that the bacillus coli communis is a very, very frequent inhabitant of the gall-bladder; and I should want strong evidence to show that a peritonitis in such a case was sterile, although it is only fair to add that some experimenters claim to have produced sterile peritonitis experimentally in rabbits by injection of sterile bile. Bearing on this question you may remember that cystic adenomata of the ovary may rupture into the peritoneal cavity. Grafted on to

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the peritoneum are adenomatous growths. In such cases often there is no general peritonitis, and it looks as if that were an instance of sterile irritation of the peritoneum without resulting general peritonitis. The last point in connection with this subject, which is worth mentioning to you, is that it has been said that sometimes the fluid in the peritonitis which occurs after strangulated hernia is proved to be bacteriologically sterile. If that is so we must suppose that the peritonitis was set up by the toxins which were transmitted through the gut. So I want to impress upon you that the result of all this is that clinically we hardly know of any peritonitis that is not due to micro-organisms, and that clinically by far the most important cause of the introduction of these micro-organisms into the peritoneal cavity is perforation of a viscus, and the next most important cause is entry of organisms through the female genital tract. If you now have these facts deeply grafted in your minds you will have done a good afternoon's work.

I do not pretend that this is a systematic lecture, so I do not propose to give you all the symptoms; but I think I might do you some service if I directed your attention to some important points about the symptoms which may help you. All the books try, and often with great success, to give a graphic description of a patient suffering from peritonitis. The first thing that every book tries to give you is—and quite correctly, and this is very important for you to get hold of—that it is in very nearly all cases a disease of sudden onset. The patient is suddenly taken ill. A woman may wake up in the morning quite well, and by the evening may be at death's door. So that the great thing to grasp in your minds is that peritonitis, certainly in almost all perforative cases, is a condition which quickly brings the patient to the edge of the grave, or passes her into it. With regard to the aspect of the patient, that is very important. Text-books give you a graphic and well-painted account of the appearance of the patient; the sunken eyes, the drawn features, the livid and yet pallid complexion, the look of anxiety; altogether the sort of face that the practised physician well knows shows what is the matter with the patient. I admit that in the majority of these cases it is present, it is very striking, and it is important; but you will commit a grave error if you go away with the impression that it is always present. You must not refuse to diagnose peritonitis because the characteristic look is absent. You may see a person who is only a few hours' from death looking calm and comfortable; there may be nothing whatever in his appearance to attract your particular attention.

There are few diseases in which more is to be gained by simply looking at the patient. The physical examination in such cases helps little compared with looking not only at the face, but turn down the bed-clothes and look at the patient. The first thing that will occur to you is that, as the books tell you, the abdomen does not move on respiration. That is very true indeed; as a rule the abdomen is perfectly immobile, and that fact is most important for diagnosis. But occasionally it may move a little, and then you may sometimes hear a rub over it from the inflamed surface rubbing against the inflamed parietes. Another thing which is very important is, do not be carried away by want of distension. I admit that the usual sequence of events, after a perforation has appeared, is that the abdomen becomes at first retracted and the muscles hard and rigid as though they were contracting down upon the perforation and trying to compress and seal it. But usually that passes away, the intestines become paralytically distended, gas accumulates in them, and consequently the abdominal distension is enormous. Do not go away under the impression that the rigidity and retraction of the abdomen always pass away. I have seen a patient die before any distension occurred. You ought to regard persistently rigid retraction of the abdomen as quite as important as distension, and remember that sometimes it is not succeeded by distension. Of course, you know the importance of observing whether the legs are drawn up. Also, in order to help the muscles of respiration, as the breathing is done by the thorax alone, the patient usually lies in bed with her arms thrown back on the pillow. The tenderness in these cases is often very extreme. If you go into the wards where there is a patient suffering from acute peritonitis, and who has not yet been operated upon, you often see a cradle over the bed. There is extreme tenderness and cutaneous hyperæsthesia with it. The pulse is a matter which it is very important to remember. It is usually a rapid, small, thready or wiry pulse, becoming more rapid and smaller and smaller as the case goes on, until when the patient is about

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to die, the pulse becomes almost uncountable at the wrist, and it can hardly be felt at all.

I have already pointed out to you that the respiration is shallow, and as the abdomen is rigid it is rather a rapid respiration. It is important to note that. The temperature does not help you very much, for this reason: the first effect of the perforation, the shock, is liable to depress the temperature; and so when you first see the patient he is collapsed, and it is only when the period of reaction sets in that the temperature mounts to the normal. Then, as a rule, it gets well above the normal. But the exceptions are so numerous that you cannot depend much upon the temperature to help you in the matter; but it is true that if it is very much raised or very much depressed, those two facts will help you. The first reaction from the collapse follows the induction of the toxæmia, due to the absorption from the abdominal cavity. The patient frequently vomits, and it is an important point to remember that the vomiting naturally tends to stop. But that does not mean that the patient is better. Even if the vomiting stops, the patient often continues to retch, and towards the end he will bring up brown fæcal-smelling matter. A common cause of death in these cases is for the fluid which is regurgitated to get into the lungs and kill the patient.

Next, do not go away with the impression that these patients are always constipated. There may be considerable diarrhœa present, and that is an evidence of the degree of toxæmia there is in the case. Patients may also complain of difficulty of micturition. Another important thing to remember is that, unfortunately for the patient, his mind usually remains clear until the last minute before death. Nowadays you would rightly attach much importance to the examination of the blood. There are few diseases except pneumonia in which leucocytosis is more rapid than it is in acute general peritonitis. Counts showing as many as 100,000 leucocytes to the cubic millimètre have been recorded, and the increase is chiefly in the polymorpho-nuclear leucocytes. It is a matter of very great importance to take a blood count, for this reason. You know that typhoid is one of the specific fevers which does not lead to leucocytosis; so if you have a typhoid case and there is sudden leucocytosis, it is a considerable point in favour of the view that some perforation has taken place.

Those are the symptoms which I wanted to call your attention to. And other practical points in connection with it are these. As Mr. Symonds pointed out in a lecture he recently gave, a circumstance which, especially to the surgeon, is of great importance, is as follows:—The first thing which occurs after perforation is great shock. The patients become very suddenly ill, that is to say, within a few hours. But they make an attempt to rally, and, compared with their serious condition, they may, at the end of twenty-four hours, feel so improved that they themselves declare that they are very much better. A period of reaction or repose, as the patient calls it, sets in, which, compared with the previous collapsed state, is apparently very satisfactory. But that is really a most dangerous period, because the unwary are inclined to say that the patient is really better, and, consequently, they do nothing. When you have a case of that sort before you be very careful to see whether the *local* signs are any better. That is to say, is there still retraction, is there distension, is there local tenderness, is there rigidity? It is by the local signs you will be able to tell whether the period of reaction or repose indicates that after all it was only a serious attack of colic and not a perforative peritonitis which you had to deal with. If you see the patient in that period of repose and you fail to give it its proper significance you may say I will not operate, and you may come next morning and find the patient dead or dying.

The other point I wanted to direct your attention to is, that in certain patients acute peritonitis gives no symptoms at all. When a man is all but dead you cannot expect many symptoms to be manifested in him. Symptoms indicate a certain amount of reaction; if the poor patient is moribund he cannot feel pain, and so it is not infrequent in acute peritonitis to miss the real trouble. I remember missing it, for instance, in a case of malignant disease of the bowel. Perforation occurred, and the patient died of peritonitis, but he was almost moribund from the malignant disease. He complained of no pain, and had no symptom pointing to peritonitis. I have known the same to occur in typhoid fever.

The duration of peritonitis varies very much. I have here a record of the duration of seventy-seven fatal cases of peritonitis collected by Treves which I thought might interest you, as showing

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how rapidly the subjects of this disease die. He found that in fourteen death occurred within thirty-six hours; in six death occurred within thirty-six to forty-eight hours; and in twenty death occurred between the third and fifth day. That is to say, in nearly half of them, people, often previously apparently healthy, were dead on the fifth day. In 33 per cent. death took place between the fifth and seventh days, and 27 per cent. with the seventh day. That will give you some idea of the severity of the disease with which we have to deal.

The prognosis is very grave indeed. The outlook for such cases is improving, because surgery is improving. It is generally said to have a mortality of about 80 per cent., but that very likely understates the case, because it is possible that some had not got it who were thought to have it, or the peritonitis was local. In perforative peritonitis, the prognosis, apart from operation, is almost hopeless. In the cases which spread from the female genital organs, the prognosis is a little better. Not long ago I saw a case of gonorrhœal peritonitis get perfectly well without any operation.

Another thing which troubles you much when you are called to a case is the diagnosis. I do not believe in writing out lists of things by which you can tell the diagnosis of one disease from another. I believe that the best way to prevent mistakes is to be aware of the possibilities of the errors which you are likely to make. Therefore I have jotted down for you the things which I have known acute peritonitis to be confounded with. They are:—intestinal obstruction, intestinal colic, hepatic or biliary colic, renal colic, lead colic, cholera, dysentery, ulcerative or other varieties of colitis, acute pancreatitis, twisting of ovarian pedicle, pleurisy, and pneumonia. All those things I have seen confounded with peritonitis. I think if, when you see before you a case of acute peritonitis, you bear that list in mind you will be much more likely to avoid making mistakes than if you merely get up a list of distinctions.

The next thing to pass on to is the treatment. In the great majority of cases absolutely the only thing to do is to hand the patient over to a surgeon, to ask him to open the abdomen and deal with the perforation. As far as we know, perforative peritonitis never gets well apart from interference by the surgeon. It is essentially a condition in which time is everything. An hour saved adds greatly to the chances of the patient's recovery, so that it is a condition in which you must act promptly. That is why so few of you ever see a case in the acute stage; by the time you get into the wards, the patient has been operated upon. But to many of you it will often happen that for some reason or another the patient cannot be operated upon. There are probably many occasions in which, in some out of the way part of the world, there may be no facilities for operating upon a patient; or, again, the patient may refuse to be operated upon; or, as unfortunately sometimes happens, the patient is too ill for the surgeon to care to operate. He says, not unnaturally, that the operation will only hurry the end. So we must think of the treatment in cases where the operation cannot be or is not performed. I should have added that another reason why operation is not performed is that in some of the milder cases following upon infection through the female genital organs, surgical interference is not called for. In the interesting case of gonorrhœal peritonitis which I have referred to, the patient was not ill enough to warrant operative procedure being suggested, and she got quite well. So supposing that for some reason or another the case belongs to the few for whom no operation is required or cannot be had, what shall we do? Of course it is absolutely essential that the patient should be put to bed, and kept on the back, quite at rest, and there must be a cradle for the bed clothes to rest upon to keep their weight off the abdomen, and the woman should have a pillow under her feet so as to rest her legs when they are flexed up on the abdomen, that being the position which is most comfortable for her.

It is a short illness; the patient will probably either die or be much improved by the end of a week or so. So that the quantity of food is not a matter of very great importance. Rest to the abdomen is more important than food. I have pointed out to you that such patients are generally sick at the beginning of the illness, and that they suffer much from nausea and retching. So you will usually give very little or nothing by the mouth, and you will therefore have to feed by the rectum. You want to feed the patient as rarely as possible, so that you may disturb her as little as you can. Thus large enemata are better to use than small ones, and those mentioned by Dr.

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Beddard are excellent. For example, somatose 3½ oz., dextrose (5·8 per cent. solution) 9 oz. is a good enema. Another made of 9 fluid oz. starch and water, with the white of three eggs, with 40 grs. of salt to the 9 oz. is good. Do not use nutrient suppositories, because they have little nourishment in them. You cannot often move a patient with acute peritonitis; but when you have passed the soft tube high up, try and tilt the patient upwards, or raise the end of the bed, so as to get the enema to flow a long way up. And then you will be much more likely to get the enema absorbed if you first of all wash out the bowel with a little saline solution. There is another point which is not sufficiently thought of. These patients are very sick, so that they quickly lose much fluid, and hence one reason, I think, why many of them are as ill as they are is because they have not got enough fluid in them. So you should do one of two things; either some time when you are not giving your enema throw a quantity of normal saline fluid into the bowel, or transfuse a salt solution into the cellular tissue about the axilla or under the breast. Do not let the patient die for want of fluid.

The mention of vomiting reminds me of the treatment of that. It is very difficult to treat it. Fortunately it often stops of itself. Of drugs, I think prussic acid in a little effervescing mixture is good, or drop doses of tincture of iodine. But I am sure that often vomiting may be relieved by washing out the stomach. As a rule, men are afraid to do that in cases of peritonitis; they say surely it will make matters worse. But if you let these patients go on vomiting they will die, and I have not seen any harm follow washing out of the stomach provided there is not a hole in it, and by that means you are able to get rid of the filthy brown stuff which, if absorbed by the stomach, must be very bad for the patient. Do not forget that sometimes vomiting is due to the opium which you are giving. Another matter is the giving of ice to these people. Many of these patients are allowed to be constantly taking up ice to suck. Think of the loss of energy that the melting of ice means. Every time the patient gets a piece of ice and sucks it there is so much heat abstracted from the body. Cut down the ice as much as you can; there is no real reason why they should have it at all. Certainly the less they have the better for them. You will try to keep the mouth clean by tincture of myrrh and borax.

I have left to the last the question of the giving of opium. Surgeons frequently object to the giving of opium, and say they wish it had not been done. And they are quite right, because I have pointed out to you that the majority of cases of acute peritonitis will pass to the care of the surgeon and require laparotomy because of the peritonitis, but the opium has naturally dulled the symptoms, and so by the time the surgeon sees the patient he is less likely to diagnose whether there has been perforation or not. All that is true, but I am supposing you are dealing with one of those few cases in which there is no perforation, in which there is no operation going to be done. If there is no operative interference contemplated you must thoroughly push opium, for this is your best chance of saving your patient. The gain which you have by that means is immense. You paralyse the bowel and keep it still, and keep the inflamed parts at rest, and thus enable adhesions to form and for recovery to take place. The patient should be kept gently under the influence of opium. And when you are observing whether or not you have given enough opium, do not direct your attention chiefly to the respiratory movements, because owing to the peritonitis the respiratory movements are less. So you may think you have given enough opium when in reality you have not. In these abdominal cases the guide as to the quantity of opium is not the abdominal movement, but the state of the pupil. The best way to give the opium is to put the requisite quantity of tincture of opium into the enema which you are giving, and give such a quantity that the patient is kept under its influence, so that he is always a little drowsy. If you will treat a case which for any reason cannot be operated upon like this, you will find that fortunately it may sometimes get well.

Sympathectomy in Exophthalmic Goitre.—Balacescu reports seventeen cases of exophthalmic goitre treated by total bilateral resection of the cervical sympathetic. The results are ten cured, two cured but with recurrence since, and five improved. The patients have been under observation twenty days to two years. He considers it comparatively harmless, and the most effective of all operative methods of treating this disease.—*Journ. A. M. A., May 31st.*

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A CLINICAL DEMONSTRATION

At the Medical Graduates' College and Polyclinic, January 30th, 1902.

By JONATHAN HUTCHINSON, jun., F.R.C.S.

GENTLEMEN,—I have first to show you a man æt. 44, who was suffering from a fractured patella which I treated by wiring. I will ask him to bend each knee alternately, and then invite you to see if you can tell me on which knee the wiring was done before I ask him to take off his clothes. I think you will admit it is impossible to tell. His power of flexion is perfect. I have asked two of the cases of fractured patella treated by wiring in the last two years to come up and let us see the result. They are merely sample cases and in no way specially selected. I have not examined them to see whether the result as regards movements of the knee is perfect or not. I will pass round this specimen of fragments of patella, to illustrate the method of wiring. It is the only one I have obtained after operating myself. The patient was a woman who fractured one patella and was taken to the London Hospital, where the fragments were wired together. It was an unusually bad case, because it was a comminuted fracture, and a small wedge-shaped piece was loose; this I removed. The anæsthetic was carefully given by Dr. Hewitt, than whom there is no more skilful anæsthetist in London, and he noticed nothing wrong with the patient at the time. She did perfectly well for three weeks. At the end of this time when sitting up one day she was seized with sudden faintness, and died of cardiac syncope in a few minutes. At the post-mortem there was found extensive disease of the heart; there had been no bruit accompanying this, and hence it had escaped detection. The knee-joint was perfectly normal, and I dissected this patella out. Death occurred only three weeks after the wiring, yet you will see that the union is so firm that I doubt if you could pull the fragments asunder if the wires were removed. Of course the wires constitute additional strength, but there is considerable bony union present. The wires will strike you as somewhat loose, but you must remember that the specimen is a dry one, and the aponeurosis in which they were buried has shrunk up. The case is interesting as bearing upon the dangers which may arise from the anæsthetic; in such a case no one could foretell that this fatal termination would ensue. That patient was examined by Dr. Hewitt before the administration, but nothing abnormal was found, and she went through the operation satisfactorily and lived three weeks after it. The case bears out the view that even when there is heart disease a general anæsthetic may be given, though of course there is grave risk sometimes. To return to our patient, in him the operation was done in July, 1900, that is to say eighteen months ago. He is employed in office work and does not have to kneel much, so in that respect he does not furnish the best test of what a man can do after this operation of wiring. This patient says he sometimes walks about all day and feels no inconvenience from his knee. He can also easily go upstairs. Sometimes when he is walking downstairs he can hear a crackling noise in that joint. I never take out the wire in these cases unless it is producing irritation, which it hardly ever does. The only other thing to notice about the case is that there has been some slight wasting of the quadriceps extensor. Slight wasting probably occurs in most of these cases, but nothing compared to what is usual in those not wired. Here there is a difference of about three quarters of an inch in the circumference of the two thighs at their centre. You see he has absolutely perfect power of bending both knees, and he can kick as strongly with one as the other leg. In using a splint for two or three weeks in these cases perhaps one does what is really unnecessary, and some surgeons do not employ a splint at all after wiring. The operation is usually performed about ten days after the accident, when the effusion has largely subsided. Before wiring the patient is kept quiet in bed for three successive days, and on every one of those days full preparations are made just as if the operation were going to be performed then. That is to say, three times over the knee-joint is cleansed outside with an alcoholic solution of carbolic acid 1 in 20, or biniodide of mercury, or perchloride, and after the third day, provided we have meantime discovered no reason against operation, the incision is made. I do not think it matters very much whether a lateral flap is turned over, or whether a longitudinal incision is made, or a horse-shoe flap turned upwards. The joint is freely opened, the blood-clot is cleared out with sterilised

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sponges, the pieces of aponeurosis are lifted up which always hang down into the gap, and the edges of bone are thoroughly cleansed. The bone is then drilled in two places, each fragment being drilled obliquely downwards, so that the wires can be introduced just short of the cartilage. The wires ultimately do not lie in the joint or touching the cartilage, but come just short of that. Of course you must take care to get a firm grip of the fragment of bone. Grooves having been cut in the aponeurosis in which the wires can lie, the four ends are drawn together at the same time, twisted tightly, and hammered down, any sharp point which may be projecting being cut off. Then the wound is closed up. As I have said, it is probably unnecessary for any splint to be worn, but in the case of the incision being made below the patella it seems advisable to wait until the wound is healed before flexion is commenced. I have never seen synovitis follow the operation in any of my cases. I think you will agree with me that you cannot guarantee, by any other method, such a result as this patient shows. There is no other method I know of by which you can allow your patient to freely bend the knee within a month of the accident. Nearly all the conservative methods involve keeping the leg stiff and straight for about six months, or probably more. There is no other method of treating fracture of the patella by which the union is stronger than it was before, because at the end of a couple of months there is bony union between the two fragments, plus the strong wires which you used to unite the bones. The only alternative which perhaps is as good consists in using stout kangaroo tendon in place of wire. Possibly that may be as good, but I like to feel I have the added strength of the wires, as well as the true approximation. With regard to sewing the edges of aponeurosis together without drilling the bone—and all sorts of modifications of this have been introduced—I do not believe in any of them. They have no special advantage, and I have seen skiagraphs of cases treated in that way, and there is no firm bony union. So many methods which are advocated as bringing the ends together result in tilting, and then re-fracture readily occurs. I think by wiring re-fracture is not likely to occur. Of course one admits there are drawbacks to it. Now and then a case ends calamitously, and I have seen one such within the last year, at a provincial hospital, where the patient evidently was losing his life as a result of septic complications. But one would say that in properly selected cases there is no method that rivals the bringing of the portions together by silver wire. Some few years ago I received a message to go down to the London Hospital late at night to wire a fractured patella in the case of a policeman. The message was from a doctor, who said he had sent the case on. I did not go to wire the patella at once—the accident had occurred the same day—and I know I incurred comment in consequence of my supposed negligence. If I had gone to wire a fractured patella directly after the accident, without proper aseptic precautions, I should have been guilty of the most criminal negligence, and as a matter of fact the case was not a suitable one for operation. Suppose there is heart trouble, or albuminuria, or the patient is alcoholic, you have no right to submit him to an operation with considerable risk like that of wiring the patella. And you should never do that operation until you have made the part aseptic. Moreover, you should not do it until the inflammatory trouble in the knee has gone down, *i.e.* ten to fourteen days. I have been asked how long after the injury to the patella should wiring be performed. A few surgeons reserve wiring for old cases, where there has been persistent gaping and much weakness in the limb. These are, however, the worst cases to operate on, and I have declined to operate in a fair number. The fragments have been drawn apart two or three inches, months have elapsed, and the quadriceps has wasted and shortened, and the upper fragment cannot be brought down by any ordinary force in apposition to the other. Also the capsule has by this time become altered a good deal. If you have to make a wide gap in the quadriceps tendon what advantage do you gain? We grant that sometimes perfect results are obtained from non-operative treatment. One patient I saw had something like two inches of separation between the fragments. The fracture had been sustained before wiring came into fashion. In spite of his gaping patella that man had walked to Brighton from London in a day. On the other hand, I have seen a patient twelve months after transverse fracture of the patella who was still getting about on crutches, and who considered the limb practically useless, as he had very little power

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over it at all. Our second patient runs up and down ladders in his daily occupation without the slightest difficulty. The scar is not adherent to the line of fracture, and you cannot feel the wires, because they are so embedded in fibrous tissue. I do not bring these cases here as a triumph of operations done by myself; it is simply to show you what is the average result in properly wired cases. The point is of great importance with regard to the evidence which you may have to give in courts of law. You are called to a case of fractured patella, the patient doing laborious work, such as that of a builder. You are asked two or three months afterwards to estimate the damage which that man has sustained. With the non-operative treatment you will admit, I think, that the damage usually proves to be considerable. The man will rarely be as strong as he was, and in regard to many parts of his duties he will be much hampered. But you might say that with satisfactory wiring the man is as good as ever he was. Very much therefore depends upon whether the man has been operated upon or not.

The Best Form of Operation for Epithelioma of the Penis.

Here is a man æt. 46, whom I asked to come up. He is a married man, and has five children. His disease of the penis commenced nearly a year ago. It was a case of a familiar type, where the foreskin is long and retracted with difficulty, and where a certain amount of sanguineous discharge is noticed under the foreskin. When the part was examined there was felt a hard mass underneath which might be taken for an indurated chancre. But in this patient there had been no exposure to venereal disease, and I believe the correct diagnosis was made fairly early. The diagnosis was made of malignant disease of the prepuce encroaching upon the glans. I have seen several cases of epithelioma come to the Lock Hospital in which the diagnosis of indurated chancre had been previously made, and much time wasted simply because there was a hard mass to be felt under the foreskin and some sanguineous discharge came from it. You can often diagnose the complaint by examining the discharge microscopically after staining it with methylene blue or methylene violet. By that means you can generally prove that the discharge is coming from an epitheliomatous ulcer, and not from a hard chancre. In any doubtful case the prepuce should be slit up and the diagnosis thereby established. In our patient there were indurated glands in both groins. These were dissected out at the same time as the amputation. I wanted you to notice the stump left after amputation of the penis. The meatus is as good as one could wish, and I have not known any cicatricial contraction following this method of amputation, which I have performed at least twenty times. There is nothing original in it; it is simply a dorsal flap of skin, which is brought down over the surface of the corpora cavernosa and united to the skin on the under-surface of the penis. The urethra, which is cut half an inch longer than the corpora cavernosa, is brought through a small aperture in the flap, and sewn carefully all round its edges, being everted by means of two small incisions. There is then no retraction and no likelihood of stricture, and one can pass a full-sized bougie the first time the patient comes up to be examined after the operation. As to operating by any method other than by careful suture, such, for instance, as the galvanic écraseur, I believe it is dangerous and wholly unsurgical. I am also sure that in most cases of epithelioma of the penis it is unnecessary to do the very severe operation known as the Thiersch-Gould operation. In certain cases these operators advocate dissection of the corpora cavernosa from the rami of pubis and ischium, and that the urethra should be brought out in the perineum. But it is not a pleasant thing to have to micturate always with the thighs widely separated, and the only bad case of stricture I have had following amputation of the penis was one of this kind. The reasons for not adopting such a severe procedure as dissecting the corpora cavernosa from the bone and bringing the urethra into the perineum is this: one has found that epithelioma beginning at the glans penis has no special tendency to spread along the urethra; that I say with emphasis. It has no special tendency to infect the corpora cavernosa; but where it does infect and cause dangerous deposits is in the inguinal glands. If you take cases of epithelioma of the penis which end fatally you will find that such a result is not due to deposits in the corpora cavernosa, or carried up to the vena cava, or along the urethra. It is practically always in the groin. A lump forms here; the skin gets adherent over

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the femoral vein, ulceration occurs, and the patient dies exhausted of sapræmia from the foul resulting surfaces in the groin. Now and then, as a pathological curiosity, secondary deposits in the lungs or in other viscera have occurred, but that is extraordinarily rare in epithelioma of the penis. Therefore why one should subject the patient to such a severe procedure as the one I have mentioned I do not know. There is a gentleman on whom I performed this operation of amputation of the penis five years ago. He had secondary glands, which were adherent to the femoral vein and Poupart's ligament in the groin. I removed those glands when I amputated the penis, and I assure you that that man told me he had since been able to perform not only urination, but the sexual function with satisfaction to himself. That is a point which most patients will make a great deal of. In this man there is no sign of recurrence in the glands of the groin, but it is yet too early to say very much about that in his case. However, six months have elapsed, and I hope he is free from that risk. There is a point I want to mention about the free dissection out of glands in the groin. By removing the oblique set freely you destroy the lymphatic vessels, and you bring about a considerable amount of interference with the lymphatic drainage in the groin. Hence there are certain special risks which may occur. I have seen the wound exude a considerable amount of thin fluid, which no doubt was simply lymph from the divided lymphatic trunks. Union of these wounds does not compare with that of incisions made higher up, as, for example, those made in radical cure of hernia.

Epithelioma of the Tongue.

Our next patient is a man æt. 74, with a large projecting tumour of the right side of the tongue. You will at once notice that he has tremor of both arms. He has not been bedridden, and has been able to walk about fairly well. He has had this tremor fifteen years, is thin, and has lost flesh lately. Since the beginning of October, that is to say four months ago, he has had a shooting pain in the right ear, and for about a fortnight he has noticed an ulcer on the tongue. He never had any trouble with the tongue before October. He smokes one to two ounces of tobacco in a week, which is a small allowance for a working man. There is no history of syphilis. He has a lump over the top of the common carotid artery, opposite the hyoid bone. That lump one recognises at once as a much enlarged cervical gland. It is at the bifurcation of the carotid artery, and certainly adheres to the jugular vein. There is only one possible diagnosis, and that is epithelioma of the fungating form, with a secondary glandular mass in the neck. In a similar case of epithelioma in a very old man six months ago, whom I saw again this week at the London Hospital, there were enlarged glands at the bifurcation and down the carotid artery. I removed those with the submaxillary gland, tied both lingual arteries, and dissected away all the lymphatic glands I could find, leaving only one eighth of the tongue at the back. He has remained well for four or five months, and now comes with extensive recurrence on the left side in the glands, and a similar recurrence in the floor of the mouth. I am afraid one must admit that though with a free incision in the neck you can dissect the glands off the main vessels fairly satisfactorily, the cases are never hopeful; and the question which arises is, is the risk to the patient worth the slight chance of permanent relief which is afforded? I should be disposed to advise this patient not to have the operation done. There is some risk of the operation proving fatal in his case (you must remember his age is 74), and the chances of a cure are remote. At the same time if, knowing the risk, he is willing to run it, and to have the operation done, I would be quite prepared to do it. One might find it impossible to get that gland mass away without tying the common carotid; and if that be done hemiplegia would probably result. If, therefore, it were found impossible to dissect those glands away without tying the carotid I should leave them.

[*Note.*—The patient was subsequently operated on—the glands, fortunately, could be peeled off the carotid artery, and half the tongue was excised. There was singularly little bleeding or shock, and the man made a perfect recovery.]

This young woman (æt. 24) came to the London Hospital with a small tumour of the right thigh, about four inches above the knee. She is single, and when she was nineteen she had pain in the right thigh. You can feel a lump there. As the pain recurred from time to time she went to a

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doctor. He could find nothing then, and it is very easy even now to overlook the lump. Therefore we do not know whether the lump was there five years ago. The probability is it has been growing five years and longer. Five months ago she said the pain became almost continuous, and she could not walk, except with assistance of sticks. Then the doctor found this tumour, of a doubtful nature. Under the remedy which we should always fall back upon in these cases, iodide of potassium, the pain and swelling are said to have diminished. But it is worth noting that in epithelioma of the tongue and other tumours which are not syphilitic, iodide of potassium does diminish the pain for a short time. That often leads to protraction of the treatment when really the surgeon ought to be called in without delay.

Sir Frederick Treves used to point out frequently in the London Hospital that iodide of potassium diminishes the pain and swelling in epithelioma of the tongue, and gives to the ulcer the appearance of promising to heal. But that is quite a deception. In this case also I attached very little importance to the statement that under iodide of potassium the pain and swelling were reduced. Moreover, as the swelling is deep in the muscle, it is difficult to estimate what the size of it is, and the patient is apt to believe that more reduction has been effected than is really the case. I do not know whether she is hysterical, but we must accept the statement that the pain has got much worse lately. In the family there are four other children, all healthy. There have been no miscarriages, and the father and mother are said to be healthy. It is the only tumour which is present on the body. When she contracts her thigh muscle you are apt to think the tumour is adherent to the bone, and the suggestion has been made that it is so adherent. But when she relaxes the muscle you find the tumour can be moved from side to side. The leg is somewhat tender. The swelling seems to be in the vastus internus. I take it it is an intra-muscular tumour, and I do not think it has anything to do with the upper end of the knee-joint. One thinks of fibroma, of neuroma, of sarcoma, of fibro-sarcoma, of hydatid cyst, of gumma, of phantom tumour, including muscular hernia, or hernia through the sheath, and asks whether there was any severe injury or sprain coincident with the onset of pain, but the answer is negative. If one had found similar lumps along the ulnar nerves one would have suspected it was a neuro-fibroma, so called. With regard to phantom muscular tumours I remember one case in which, the fibres of the adductor longus having been torn, whenever the man sat up there was a hard, solid lump, which anyone would call a tumour in the muscle. But if the muscle was relaxed the tumour disappeared, and in that sense it was a phantom tumour. I omitted to mention that it might be a lipoma. One knows how deceptive lipomata in the muscles are. There are cases, for instance, where the leg has been amputated for lipoma under the belief that it was sarcoma, and the surgeon found, to his chagrin, that it could have been enucleated if he had cut straight down upon it. I do not see the object of this patient waiting any further. She says she has a good deal of pain from this, and if she is willing to have an exploratory incision made it is the right thing to do. Without that we can only guess as to its nature.

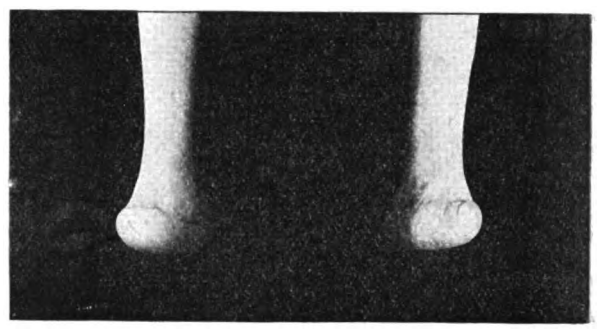

FIG. 1.

[*Note.*—On exploratory operation being done in this case the diagnosis of intra-muscular swelling proved to be correct, but it was not a true tumour. There was thrombosis of a clump of veins in the vastus internus with much cellular infiltration around it. Possibly its origin was nævoid.]

This man has just come from the London Hospital, and his statement is that for forty years he has had some disease of the foot, which commenced as a small warty growth at the upper edge, and has since slowly spread, but during the last three months its progression has been more rapid. He says he has never had syphilis. You will be struck by the odour of this lesion on his foot. It is due to decomposition between the cauliflower-

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like masses. In the tongue of the man you have seen the growth was not typically cauliflower-like in character, but this is. One could almost guarantee that under the microscope we should find squamous epithelioma, with large cell nests and extreme keratinisation. I take it he has had a warty growth on the foot for many years, and that it subsequently became epitheliomatous. He has one large gland mass in the vertical-femoral set, lying deeply, and several others are very appreciable. He has had amputation of the foot suggested, but declines. We cannot urge it owing to the extensive gland disease. Supposing, however,

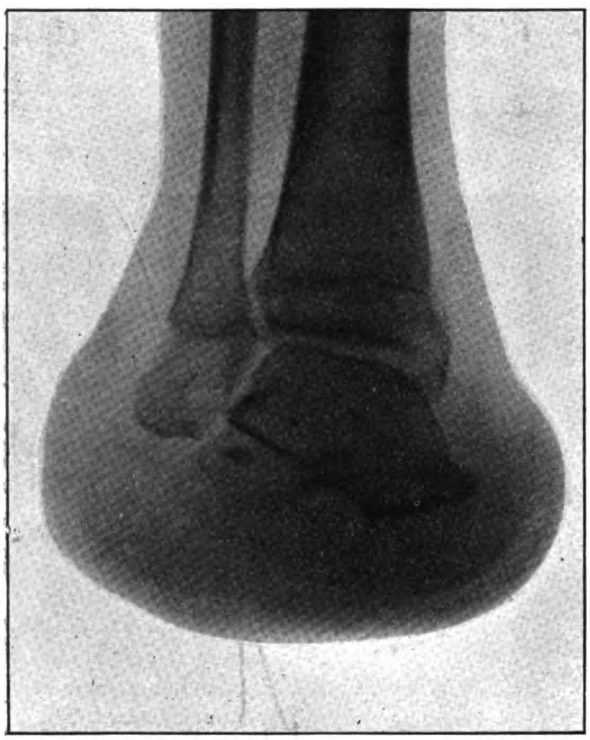

FIG. 2.

that amputation were done, perhaps a Syme would be the best, as the growth comes near to the astragalar flap. Whenever it can be done I prefer sub-astragalar amputation to a Syme; it gives a more elastic, a longer, and a better covered stump. These points are illustrated by the accompanying figures 1 and 2 from a case of sub-astragalar amputation which I performed three years ago. It will be seen from the photographs that the stump is covered with a thick pad which has not atrophied, and that the scar is well out of the way of pressure.

CANCER: ITS ORIGIN, NATURE, AND GENERAL PRINCIPLES OF TREATMENT.

(*Debate continued from page* 144.)

MR. FREDERIC EVE.—Mr. President and Gentlemen,—The causes of cancer, in my opinion, are multiple. The early stages of the disease may best be studied in the skin and in some of the superficial mucous membranes. Doubtless similar changes occur in the deeper parts, but we cannot see them. I propose to allude to some of these changes in order that we may consider how they act in determining carcinoma. These changes are well known to you, and therefore I will merely recapitulate them as rapidly as possible. In the first place, mechanical causes: the clay-pipe epithelioma of the lip, jagged teeth, leading to epithelioma of the tongue. Secondly, there are chemical causes: tar and petroleum. Tar and petroleum are much more common causes of cancer than is generally supposed. In order to illustrate this subject, I show you an epithelioma of the scrotum of a man who had worked in a tar compound for twenty years. The remarkable fact about this case was that he had simultaneously an epithelioma of the scrotum and also of the leg. Then we have various inflammatory and septic processes which give rise to cancer: leukoma, chronic inflammations of mucous membranes, chronic septic ulcers, chronic gastric ulcers, gall-stones, and, finally, balanitis. In many of these conditions one may predict that carcinoma will ultimately supervene if they are not cured or the part removed. Finally, we have granulomata, associated with tubercle and syphilis. To illustrate these conditions I will call your attention to these drawings. This one shows a warty epithelioma near the anus of a man æt. 66. It commenced twenty years before he came under observation as an oyster-shell-like growth. After removal microscopical examination showed that the surface of the growth was an epithelioma, but beneath it was a thick layer of typical tuberculous granulation tissue. The case was therefore an example of epithelioma growing on a verrucose tuberculosis of the skin. Here is another illustration of the same thing: an epithelioma springing up in lupus of the

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face of thirty years' duration. I wish particularly to draw your attention to a remarkable circumstance in regard to this case, showing what a strong determining cause the granulomatous process is, namely, that two years subsequently to the removal of the epitheliomatous ulcer the patient came to me with another epithelioma starting near the angle of the jaw absolutely distinct from the first one, the scar of which remained sound; the glands also remained sound. I have seen the same thing in a gentleman who was suffering with syphilitic leukoma and superficial ulcers of the tongue. In 1889 I removed an early epithelioma of the tip of the tongue. He remained perfectly well for nine years, and then returned with epithelioma some way back on the side of the tongue. The scar of the first growth remained perfectly sound, as did the glands, and there can be no doubt the second epithelioma formed entirely distinctly from the first and independently of it, but owing to a continuance of the same inflammatory process. I only mention these cases and these processes in order, as far as our present means of investigation extend, to examine how they act. In the first place, as a result of the irritation, we get an abundant migration of lymphocytes into the corium or mucosa, as the case may be, and they may also pass into the epithelium itself. By these means the basement membrane is broken down and the epithelium loosened from the subjacent connective tissue. Further, a layer of granulation tissue (a tissue extremely favourable for the growth of epithelium) is formed, into which the epithelium projects and grows, and it is this ingrowth which constitutes carcinoma. It may also be supposed that the irritative or inflammatory processes under consideration lower the resistance of the connective tissues to the invasion of epithelium. This occurs especially in the aged, as long ago pointed out by Thiersch. But this is not all. In all these processes, not due to a mechanical or chemical irritant, we have, in addition, either pyogenic or the specific micro-organism of syphilis or tubercle, the products of which are continuously being given off and stimulating the epithelial cells. There is therefore a continuous and abiding irritation, which causes the epithelial cells to proliferate, alters their potentialities, and causes them to grow into parts in which they do not normally exist; in fact, they assume parasitic habits. The practical bearing of the facts is the necessity of curing, if need be, by operation, of all chronic septic or specific lesions which may constitute pre-cancerous conditions.

But in many cases of carcinoma there is no element of irritation. In some of these the formation of carcinoma may be explained on the theory of "rests" or Cohnheim's theory, which is undoubtedly true as regards many innocent and some congenital malignant tumours. The difficulty in applying the theory to carcinoma is that we must assume that the "rest" remains dormant for many years before it becomes active. In order to show how long a rest may remain dormant I may mention that the other day I removed from a man æt. 62 a dermoid cyst of the rectum which had only been observed for three years. We may therefore assume that its nucleus had remained dormant for something like fifty-nine years before it became active. If this is true of dermoid cysts it may also be true of the "rests" which give rise to cancer. A good example of epithelioma originating in this manner is the cystic epithelioma of the jaw. These tumours present a striking likeness to the enamel organ; they originate from small particles or "rests" of the enamel organ which were discovered by Malassey in the alveoli of the teeth. Melanomata may also be considered as forms of carcinoma which originate from "rests." Writing ten years ago (Mansell-Moullin's 'Manual of Surgery'), I stated it as my opinion on pathologico-anatomical grounds that melanomata, or so-called melano-sarcomata of the skin, were really of the nature of carcinomata. This view is now largely held. In the great majority of cases they originate from moles. These may be considered as imperfectly developed portions of epiblast. We may therefore say that the melanomata originating in moles are derived from imperfectly differentiated epithelial cells, which, like all similar cells, possess the proliferative capacities of embryonic cells. Time permits, sir, of only touching on the fringe of this wide subject, in which conjecture far outruns fact. The cell is the carrier of malignant disease, and in its change of nature must be sought the secret of cancer. Our hope for the future appears to rest with serum therapy. Most of you must be acquainted with those extraordinary experiments * showing that

* See Dr. William Bulloch, 'Practitioner,' May, 1901.

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toxins or cytotoxins may be produced, which are fatal to or destroy various cells and tissues of the body, and these alone. If rabbits are treated with injections of bull's serum a substance is formed in the peritoneal cavity which rapidly immobilises the bull's spermatozoa. This property is not possessed by normal rabbits. The most striking of the experiments on these lines are those showing that a cytotoxin may be produced which is fatal to the various forms of epithelium. If ciliated epithelium from the trachea of an ox be introduced on two occasions into the peritoneum of a guinea-pig a substance is produced which destroys in a short time ciliated epithelium subsequently introduced into the abdomen, although in normal guinea-pigs the movements of the cilia continued for two days. When we have reached these recently explored realms of pathology it appears to be only a step to the discovery of a serum which is toxic for cancer cells. It is stated that Jansen has already done this in cancer among mice.

PROGNOSIS IN RENAL TUBERCULOSIS.

By HENRY MORRIS, F.R.C.S.

UNLESS the disease is strictly localised, and, as such, is diagnosed early enough to be radically removed, the prognosis is most unfavourable. This is especially so when both kidneys, or one kidney with its ureter and an extensive tract of the bladder, are involved. The course of the infiltrating caseous form of renal tuberculosis (as distinct from the acute miliary disease) is much more chronic than is often supposed. The disease progresses often by fits and starts, periods of activity being succeeded by long periods when it is comparatively or entirely quiescent. It is by no means unusual for the disease to extend over four or five years; its duration, in fact, varies much, from a few months to several years. Its course is hastened by the supervention of secondary pyogenic infection, and occasionally, though very rarely, by profuse hæmaturia. In certain rare hæmorrhagic cases the patients have rapidly become anæmic, and a fatal result even has been threatened by excessive hæmaturia. The complication of suppurative pyelo-nephritis is one of the gravest significance. It is not impossible, but it is rare, for this disease to subside, either by the tuberculous material undergoing cretification, or by the whole kidney atrophying owing to the complete occlusion of the ureter. Neither prognosis nor treatment, however, ought to be influenced by these improbable events. Certain well-marked cases of renal tuberculosis have been temporarily or permanently benefited, and some actually cured, by change of residence, a suitable climate, and under a suitable hygienic and dietetic *régime*. Good results of this sort are much more likely to be accomplished in the rich and well-to-do classes, by whom these advantages can be obtained to the full, than in the poorer classes of patients. The prognosis of surgical treatment has greatly improved with operative experience during the last fifteen years. The mortality after nephrectomy had been reduced from 40 per cent. when Gross published his statistics in 1885, and 38·18 according to Palet's and to Vigneron's figures in 1891 and 1893 respectively, to 18 per cent., as recorded by Israel in 1896, and 7 per cent. by Tilden Brown in 1898.

The above is taken from the work recently published in two volumes for Mr. Henry Morris by Cassell & Co., entitled SURGICAL DISEASES OF THE KIDNEY AND URETER. The first thing that strikes the reader after perusing this book is that it combines in so admirable a manner the most precise reasoning with an unique experience of the subjects discussed, that the work must be accepted as an extremely valuable and scientific addition to the medical literature of the present time. The abundant fulness of knowledge displayed by the author, and the great accuracy in regard to minute details, places this production in the very first rank, and marks it out as the most practical and exhaustive treatise on the subject. The two coloured plates and the illustrations, numbering over two hundred, are well executed, and in themselves form a presentment of the subject matter from a modern standpoint that is both interesting and instructive. We note that the author in his preface says that nearly all the illustrations are original, and for the most part drawn especially for this work. Mr. Morris also acknowledges in the preface his indebtedness to Professor Arthur Robinson, of King's College, for the great care and trouble he has taken in verifying and correcting the descriptions of the surgical anatomy of the kidney and ureter, and this portion of the book certainly will appeal to all lovers of the scientific study of anatomy. The medical profession will appreciate the labour and study expended on these volumes, characterised as they are by lucidity of expression and simplicity of arrangement.

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THE CLINICAL JOURNAL,

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CARCINOMA FROM THE CLINICAL POINT OF VIEW.*

By J. F. PAYNE, M.D., F.R.C.P.

THE study of cancer during the last half-century has been mainly histological. We now know precisely what we mean by carcinoma, by sarcoma, and by simple tissue tumours, and, allowing for a few puzzling specimens, have no difficulty in discriminating them. The time seems now come to study the subject from other points of view. The first attempt at advance made in the study of cancer, as of other branches of pathology, has been the introduction of the experimental method. But the results with regard to cancer at present achieved by this method are admitted to be very unsatisfactory.

I propose, therefore, leaving histology and experiment on one side, to consider the subject from the clinical point of view, or, more precisely, from what I call the natural history point of view. To take phenomena, of which the cause is not known, to put them together and see what hypothesis they suggest, and further to state these hypotheses, even if quite unproved, clearly and definitely, so that we see precisely what is involved in them if they are proved, and what points have to be investigated, in order to test their validity; this is a legitimate and often a far from useless method of enquiry.

As a preliminary, I would say that I speak only of carcinoma, epithelial cancer in the wide sense, not of sarcoma or of simple tumours. I cannot agree with the opinion which has been expressed that the explanation of carcinoma must necessarily be the explanation of sarcoma and the rest. It is

* The closing address of the Debate on Cancer; its Origin, Nature, and General Principles of Treatment, held under the auspices of the Chelsea Clinical Society.

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possible that the explanations of these several structures and their causes may be totally different.

Some years ago there was a great controversy or debate at the Pathological Society on the question whether cancer was to be regarded as a local or a constitutional disease. Our late honoured friend and master, Sir James Paget, defended the "constitutional" theory, affirming that cancer was a constitutional disease of which the local growths were to be regarded as the consequence, or figuratively as the expression.

But this controversy is now a little out of date. We know that a disease may be both constitutional and local. This truth has been established by the study of specific disease, especially those of which tissue changes are an important part, namely infective granulomas, with some others. We now see that a local tissue change is the beginning of a general, that is, a so-called constitutional disease, causing a general infection of the whole body. And this happens because the local tissue change is produced by an infective cause, viz. a microbe, which also is capable of producing general changes or diffuse poisoning through the whole body. It is admitted that tubercle, for instance, is both a local and a constitutional disease. But we do not admit that there is a general constitutional state or tubercular diathesis which gives rise to the local tubercles.

The question thus raised was not, however, without importance, because it drew attention to the truth that cancer, besides being on its anatomical side a growth, and thus comparable to the simple tissue growths or tumours, has also another side, that, namely, by which it is comparable to specific infective diseases. This dual character of cancer should not be forgotten.

The triumphs of the histological school have strongly emphasised the structural characters of cancer, that is, its relation to tumours, and have, perhaps, led pathologists to overlook, or at least inadequately to appreciate, the other side, that is, cancer as a disease.

Recently this side of the subject has been more studied. Many pathologists see clearly and have forcibly dwelt upon the analogies between cancer and specific infective diseases, more especially those in which there is a large amount of tissue change, such as tubercle, syphilis, leprosy, and actinomycosis.

We must consider for a moment what this view of the matter really amounts to—what do we mean by a specific disease?

We mean a chain of morbid processes which begin with the introduction into the body of some specific cause of disease, viz. a parasitic organism, generally vegetable, sometimes animal; and, if the disease is to have a continued existence, includes the passage of this organism out of the body passively or actively, in such a way as to continue its species.

1. This specific organism may produce evident changes, called the *initial lesion*, at the point where it enters the body, or it may produce none at all. There is an indefinite gradation of local effects at the point of entrance.

2. Once introduced into the body it may limit its activity to the point where it entered, or it may spread in the neighbourhood, or it may be, by various mechanical means, transported to another part of the body, where it produces effects similar to, though not always identical with, those of the initial lesion.

3. It may also produce, by the products of its local processes, a general poisoning or intoxication of the whole body, which again, according to chemical and physiological laws, will affect some parts or systems more than others.

This general intoxication may be absent, or may be a very small part of the disease. It may, in other cases, constitute nearly the whole of the disease as we see it.

There are, then, three factors in such a disease —the initial lesion, secondary local lesions, and general intoxication. The relative amount and relative importance of these factors may vary almost indefinitely.

Other evidence of specific infection is the communication of the disease to other individuals by direct contagion, inoculation, inhalation, etc., or by indirect means; and this is really still the chief evidence of the specific character of certain diseases, of which the specific microbes are still unknown, such as several of the exanthemata.

Another line of evidence is the distribution of the disease in space, that is, its limitation to certain localities. It was on this ground that malaria was inferred to be a specific disease before its actual cause was known.

But all these various lines of evidence are weak

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compared with the actual discovery of the specific cause. When the specific pathogenic microbe has been isolated and studied, all the other characters are seen to fall into their places, being explained as various means by which the microbe is disseminated through the body or in the outer world; and what is important to remember, the means by which the microbe propagates its race.

Now if cancer is to be regarded as a specific disease, the most convincing argument would be the discovery of its microbe and the proof by experiment that it can produce the phenomenon, and conform to the observed laws of the disease. But while this crucial proof is still wanting, there may possibly be evidence such as was available and held to be adequate in the known specific bacterial diseases before the discovery of their microbes, and such as we still have to be contented with in the case of most of the acute febrile diseases. Evidence of this kind would justify a certain presumption that cancer also is a specific disease.

Such evidence, if forthcoming, would fall under three heads:

1. Evidence of an initial lesion, of secondary lesions arising from this, and of general intoxication.

2. Evidence of communication of the disease from one human being or animal to another, whether of the same species or not, by contagion, inoculation, or any experimental method.

3. Evidence of a specific, local, or geographical distribution, such as has been demonstrated, for instance, in malaria.

In regard to the *evidence of an initial lesion* it is quite clear, and has been abundantly demonstrated by histologists, that cancer is in the beginning a local process. It has not, however, been sufficiently considered that in a very large proportion of cases, the primary seat of cancer has many of the characters of the initial lesion of a specific disease. On this point I propose to offer some evidence, but defer it for the present, as it is connected with another part of my subject, the local distribution of cancer in the body.

As, however, I wish to present both sides of the question, I will just draw attention to certain unmistakable differences between the initial lesion of cancer and the initial lesions of those specific bacterial diseases which produce definite tissue changes. Those which do not produce such changes need not be regarded.

The difference, broadly, is that the primary focus of disease in cancer is a growth, and the primary focus in tubercle and the like is what is called inflammation.

That tubercle and all the granulomas are really inflammations is perhaps hard to grasp. The essential point is that what is called proliferation of cells in these formations is of one kind of tissue only, that is, the connective, and is of the nature of a defensive reaction, neutralising and killing the bacteria, or tending to do so. The body resents the invasion of the microbe as an injury, and meets it, as in other cases, with a defensive reaction. The difference between the production of tubercle and that of an ordinary or diffuse inflammation is that in the former the irritant is very small, fixed to one spot, and exerts its influence over a very limited area only. Whereas in common inflammation the irritant is either diffuse or multiple, it may spread indefinitely, and hence the area affected by it is not definitely limited. The form and structure of a tubercle may be precisely reproduced by a very small mechanical irritant, such as the hair of a mouse in Baumgarten's experiments, or the hair of a caterpillar in the affection called *ophthalmia nodosa*.

On the other hand, the initial process in cancer or tumour formation is a growth. There is really no sign of injury, and the signs of defensive reaction, called inflammation, are slight or none at first, becoming marked only when the growth becomes so considerable as to exert mechanical or chemical irritation on surrounding parts. For these reasons it does not seem probable that cancer is produced by a bacterium; and we can readily accept the negative evidence which has been collected on this point. The same objections do not apply to the hypothesis on an animal, *i. e.* protozoan parasite, since some species of protozoa are well known to live as parasites within epithelial cells in some animals.

Again, the process of forming secondary foci is different. In tubercle and the like a conveyance of the bacillus from one part to another is sufficient. In the case of cancer there is a conveyance or metastasis of cells. This point also I must pass over rapidly, though it has been disputed, some pathologists having maintained that the actual cells are not transported, but only something which they call a "*seminium*." The histological evi-

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dence is, to my mind, conclusive in the direction which I have indicated, though I must pass over it now.

If this be so, we must ask what is it that the transported cells carry with them which enables them to go on growing and proliferating in the same manner as the primary growth.

Is it merely a changed mode of growth, a metaplasia, impressed upon the cells of the primary cancer by its unknown cause, and transmitted from them to their successors?

Is it some small portion of a biological "ferment" or other substance, capable of affecting the mode of growth. Or is it after all a parasitic organism, which, unlike bacteria, requires to be enclosed within a cell for transmission from one part to another?

However these questions may be answered, the fact remains that cancer has a primary seat, which I call provisionally the initial lesion, and secondary seats, which are set up by the conveyance of something from the primary seat.

We can also see that there is a general intoxication. The cancerous cachexia, at one time supposed to be evidence of a constitutional disease, is clearly secondary to the absorption of something from the cancer itself. So far it agrees with the specific diseases in which local changes produce poisonous products.

Again, in specific diseases, for the most part, introduction of the specific organism confers, at least for a time, immunity on the body against the attacks of the same organism. This is strikingly the case with cancer. Multiple primary cancers are almost unknown. That is, when cancer is once established, no new cancer can be acquired. This rule, though not quite absolute, is as much so as in most specific diseases, so far as we can conclude without experiment.

Pursuing the attempt to describe cancer as a specific disease, we next consider the question of *transmission*.

It may be taken for granted that cancer is not as a rule a contagious disease, that is, it does not generally spread from an affected person to those around.

The exceptions are few in number. Infection from husband to wife, or *vice versâ*, is thought to have been observed in a very few cases. There must be innumerable cases in which such transmission does not occur, when one or the other partner is cancerous. Artificial infection by inoculation or injection, etc., has, as is well known, been tried in a very large number of experiments by various pathologists. I will not attempt to relate or criticise these experiments. The results have been on the whole, it must be admitted, unsuccessful.

All this does not show that cancer is not *communicable* from one person to another, as are, for instance, thread worms and other entozoa, directly or indirectly, though these are not generally called contagious diseases.

I now propose to test the hypothesis of cancer as a specific disease by considering whether the modes of its supposed entrance into the human body justify such a hypothesis.

The situations of primary cancer are partly external, partly internal.

External cancers beginning in the skin are either epithelioma starting with the epidermis, or rodent cancer beginning in the skin glands, or possibly other inversions of the epidermis, namely, the hair sheaths.

The invasion of the skin is very often preceded by so-called chronic irritation, better called prolonged slight injury. This is traceable in cancer of the scrotum in chimney-sweeps, in the cancer of petroleum workers, in cancer of the lower lip believed to be produced or favoured by smoking clay pipes, and other similar cases. It is to be noted that chimney-sweeps, the class most liable to external cancer, have a high rate of mortality from internal cancer also.

It is evident that if there is any infective cause of cancer the skin is one of the channels by which it invades the body.

There is another possible channel of invasion, namely, by the internal surface of the body, that is, especially by the alimentary tract.

If there is such a means of infection it may take place through some kind of food.

We may regard as possibly caused by such an infection cancer of the tongue, the œsophagus, the stomach, the intestinal tract generally, including the rectum. The process starts apparently always in the glands, though it is not easy to be quite sure that it may not begin in the investing epithelium. We must also include a large number of cases of cancer of the liver, which is rarely primary, being

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secondary for the most part, to cancer of some part of the digestive tract, though in some cases derived from cancer of other abdominal or pelvic organs.

With these parts should, I think, strictly speaking, be reckoned cancer of the lower lip, which has an intimate relation to the taking of food, and is constantly liable to be moistened with saliva. It is much more than the upper lip, a part of the mouth. Perhaps this is the explanation of the well-known fact that it is the lower lip, not the upper, which is usually attacked by cancer. The glands of the alimentary tract, the parotid and pancreas, and also the gall-bladder, must be classified with digestive organs.

We must also consider the possibility of infection through the respiratory channels, which in many infective diseases form an important medium of infection. But in cancer this has almost no importance. Primary cancer of the lung is almost unknown, and when any kind of malignant growth is found there, it is either of internal origin from the bronchial glands or mediastinum, or else secondary, being brought by the blood from some other part. The only definite cancer of the respiratory organs is that of the larynx, and from the close connection of this organ with the mouth and pharynx, it is probable that if there is any infection it proceeds from the alimentary tract.

We should conclude that if there is an infective cause of cancer it does not pass through the air.

There remains a large class of internal cancers, of which the possible connection with external infection cannot be traced, viz. those of internal glands, such as kidney, and especially of the reproductive organs in both sexes, including the mammary gland. Malignant growths of the nervous and osseous systems are, broadly speaking, sarcomas, and, therefore, though malignant, very probably of different origin to carcinoma. But it is important to remember that cancers of glands or organs unconnected with the digestive system form a very small proportion of the whole in the male sex; and if cancer of uterus and mamma are excluded, an equally small proportion in the female.

In estimating cancer of the skin the statistics of mortality help us little, for these cancers are rarely the immediate cause of death. In the latest returns giving the numbers of cancers of different parts of the body causing death in England and Wales, I found only 574 deaths from cancer of the skin out of more than 25,000 of cancers of all parts, giving a proportion of about 2·3 per cent. in which death was actually ascribed to cancer of the skin. Of course this does not represent the real proportion, as many external cancers are presumably cured by operation, while when death results it is often set down to secondary cancer of the lymphatic glands or of some internal organ, as being the immediate cause of death.

Statistics of living patients give a very different proportion. Taking the cases of cancer among the patients of St. Thomas's Hospital for twenty years, amounting to 4291 cases, I find 695 are described as *external*, which would give 16·4 per cent. of the whole. This included some cases of sarcoma, but probably the greater number were epithelioma or cancer of the skin glands. It is difficult to say whether this represents the true proportion in the population, since in hospital practice cases suitable for operation predominate, and probably therefore this proportion is too high.

Mr. Roger Williams, in an analysis of 7297 hospital cases of cancer, gives the percentage of primary cancer of the skin in males at 14·3 per cent., and in females at 4·1 per cent. But cancers of the external genitalia really belong to the skin; these he reckons at 6·8 per cent. in males, 3·4 per cent. in women.

Broadly, we may say that something like 16 per cent., or, say, less than one-sixth of all cancers, begin on the external surface of the body.

If we now turn to the internal surface of the body, that is the whole alimentary tract, we obtain a very different result. In the statistics of mortality from cancer in England and Wales for 1899 we find 26,325 deaths attributed to cancer. In 1224 the part of the body affected is not stated, leaving 25,101 referred to different parts. Out of these no less than 14,021, or 7011 males, 7010 females, are referred to the digestive organs in a wide sense, including the liver and other glands and also the peritoneum. It may be thought that the peritoneum and mesentery should not be wholly included, since while cancer of these parts is mostly secondary to cancer of the digestive organs, it may be connected with the uterine organs. But the deduction necessary on this account would not be large.

Expressed in percentages, this gives us 56 per

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cent. of the total mortality due to cancer of the digestive organs, viz. 16 per cent. to cancer of the great glands, and 40 per cent. cancer of the external surface.

We arrive then at the remarkable conclusion that not less than 56 per cent. of fatal cancers begin on the internal surface of the body, and supposing that nearly 16 per cent. affect the external surface, in the male sex, 70 per cent. of the cases of cancer actually begin on a surface internal or external—a fact which is entirely consistent with an internal infection by something introduced with the food or by actual contact with the outer surface. In the female sex, nearly the same proportion holds if we exclude cancers of the reproductive organs and mamma, of which I speak separately.

If we regard cancer in the male sex only, at least 70 per cent. (or in some countries 90 per cent.) are due to cancer of the digestive organs.

Now primary cancer of the digestive organs is, as I have said, nearly always a surface lesion. It very rarely begins in the parenchyma of any solid organ. Hence, if there is any invasion of the body by the unknown cause of cancer, all these primary cancers of the internal surface may be regarded, like external cancers, as the *initial lesion* of the specific disease.

I have said that the distribution of primary cancer in the male sex generally, and in the female sex, if we leave out cancer of the reproductive system, points to the conclusion that it is due to a germ of some kind introduced from without, entering the body in most cases through the alimentary tract, and therefore probably introduced with *ingesta*, though in some cases through the skin.

This explanation could not apply to uterine and mammary cancer; which must therefore be considered separately.

On this point I would first observe that many pathogenic microbes which commonly produce an initial lesion may enter the body without doing so. For instance, the tubercle bacillus may penetrate the mucous membrane of the throat and lodge in the cervical lymphatic glands without any obvious initial lesion. It may even enter the blood and set up tuberculosis of joints or kidneys, without any other discoverable primary lesion.

We must also remember the universal biological law that a parasite which has its accustomed mode of entrance to the body, also has its accustomed method of exit.

If we make the hypothesis that the germs of cancer may get into the blood, we should expect to find some arrangement for expelling them, more or less successfully, into the outside world, an operation which may be for the good of both parasite and host.

Now in the female body there is from puberty till the climacteric a profuse periodical discharge of blood through the uterus. I propose the hypothesis that cancer germs may be thus eliminated, and that if this is done speedily and completely no cancer results, but if elimination be incomplete and cancer germs are left behind in the uterine tissues, a cancer growth may occur. If we look at Dr. Tatham's tables of age distribution, we see a sudden increase in the mortality from cancer after forty-five, about the time when menstruation ceases. It is as if menstruation had up to that age conferred an immunity.

Another confirmation of this theory of uterine cancer may be found in the growth called *deciduoma malignum*, which appears to me to be histologically a carcinoma, though some pathologists regard it as a sarcoma, of which we may conceive the germ to be extruded with uterine blood, and not being able to escape to set up a growth.

Should it be asked whether there is any other instance of an "emunctory" or habitual discharge conferring immunity against cancer, or its stoppage favouring that disease, I may refer to what is regarded by some as a mere superstition, viz. the apparent incompatibility of an open ulcer of the leg with internal cancer. It is a popular belief that such an ulcer protects against cancer. The first medical statement about this belief was in London at the end of the eighteenth century, when it was proposed as a subject for inquiry. I first came across the statement in Broca's "Traité des Tumeurs." Having had the opportunity in Skin Hospitals, of at least inspecting a large number of such ulcers, I have never found one co-existing with any kind of cancer. Finally, Mr. Roger Williams, in his very large series of statistics, finds the coincidence of ulcer of the leg with cancer of the breast exceedingly rare, though the patients liable to those two diseases are much of the same class and constitution.

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It is, of course, well known that epithelioma occasionally supervenes on simple ulcer, but this is rather in favour of the elimination theory than against it.

Let us meditate one other little fact which may possibly have something to do with the question. The earliest recorded increase of cancer in modern times began about the time that blood-letting began to be generally disused throughout Europe, namely, after 1840.

Mammary Cancer.—This form of cancer seems at first sight so unlike any other that it is difficult to find any analogue to it. But even in development the mammary gland is only a highly specialised sebaceous gland. We may compare mammary cancer to those forms of rodent cancer which originate in the sebaceous glands. Without forgetting the variety of structure presented by rodent cancer, I have found some and figured one in my 'Manual of General Pathology,' which closely resemble mammary cancer. I suggest, therefore, that mammary cancer is a form of sebaceous cancer.

With regard to sebaceous cancer in general, I venture to make what may be thought a very rash speculation, namely, that it represents the excretory path by which cancer germs are eliminated from the body. If one of these from time to time is imperfectly eliminated, that is, arrested on the way, it may take root, so to speak, and form a cancer. We assume that the mammary glands, like other specialised organs, retain something of their primitive function, viz., excretion. This hypothesis involves the assumption of a special function of the sebaceous glands in relation to the excretion of microbes. I will only allude to the strong probability that this is the case with regard to bacteria, though the fact is not proved.

Supposing the germs to be uniformly distributed in the blood, it is evident that in women the great majority must fall upon the mammary gland, since the bulk of this is far greater than that of all the sebaceous glands put together, and it receives a corresponding excess of blood. The male mamma, from its small size and functional inactivity, receives comparatively little blood, and, consequently, is rarely the seat of cancer. Rodent cancer appears to be commoner in men than in women, but I state this with diffidence.

The suggestion which I make, then, with regard to the two forms of cancer predominant in the female sex, uterine and mammary, is that their special localisation is due to abortive elimination of germs, which, if arrested on the outward path, produce a primary cancer.

I cannot expect that this bare hypothesis will receive immediate assent, but I would observe that I think this is the true explanation of the difference, alluded to by Mr. Shield at the last meeting, between the mammary gland on the one hand and the salivary glands (together with kidneys, testicles, and other secreting glands) on the other hand, in the frequency of primary cancer. These other glands are not excretory and have not the particular function which I have supposed to belong to the mammary and sebaceous glands.

Broadly the hypothesis is that the primary cancers represent either an initial lesion on the surface, internal or external, where the germ enters the body; or an abortive attempt at elimination which lodges the germ at some point in the excretory path. The latter is what produces the predominant cancers of the female sex, and is of subordinate importance in the male sex.

It is quite conceivable that these may represent two species of cancer, that is, they may be produced by different germs.

Considering the evidence from geographical distribution, and assuming that there is no clear evidence of cancer being a communicable disease, it may yet have the character of a specific, if it is shown to be *endemic*, that is, to be specially prevalent in certain *places*, independent of the physical characters or morbid liability of the population. The type of an endemic or miasmatic disease is ague. Anthrax is also miasmatic as well as contagious, being acquired from the soil as well as by contagion. Plague, again, we know is not always acquired or transmitted by contagion, but has a special local distribution. Tetanus is distinctly a soil disease.

This local specificity or miasmatic character was formerly attributed to the properties of the soil, as in the case of ague. But we now know that the term soil disease is not always applicable. The local element, which keeps a disease alive or makes it endemic in a certain place, is not necessarily in the soil. It may be some species of the lower animals, as mosquitoes in the case of malaria. Soil conditions are only essential in that they

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favour the existence of these insects or other animals. The soil-element in plague appears to mean rats.

Therefore, if cancer be a specific disease of the miasmatic class, endemic in certain places, it may be either that the germs exist in the soil or water, or else in some lower animal forms—insects, worms, and the like. All that is meant by this is the hypothesis that the germ of the disease carries on its continued existence somehow in external nature, not in the human body.

Some evidence of such a mode of existence of the lower germ would be given if it were shown that those who live in particular places are more liable than the inhabitants of other places to suffer from the disease, irrespective of their differences in personal condition.

I think there is a good deal of evidence to show the local specific prevalence of cancer, and this is derived from its geographical distribution.

It is a familiar fact that cancer is commoner in some countries than in others, and in some parts of certain countries than in other parts. Some remarkable facts have been collected about the geographical distribution of cancer.

We first inquire whether there are any parts of the globe where cancer is absent or extremely rare.

It is said to be very rare, or almost unknown, in extreme northern latitudes, and less common in tropical than in temperate climates.

But the *inhabitants* of different countries differ in so many respects that we cannot ignore the personal and give a predominant place to the climatic element.

The safest generalisation which can be drawn from these rather vague data is that certain races are especially liable, that is the European races generally, and the British people among them occupy a very high, if not the highest position. The same liability attends the Anglo-Saxon race, so called, in other parts of the world.

On the other hand, Asiatic peoples show a decidedly less liability to the disease, and the same is true of the African races so far as is known.

Now, this difference may be set down to that vague influence which we call *race*, a conclusion which would be important if the heredity of cancer were clearly established. It may again, as it seems to me, with greater probability be assigned to differences in social habits, especially in diet. For it is quite certain that, broadly speaking, the Asiatic and African native races are very largely vegetarian, while European peoples, and their descendants in the new world and the southern hemisphere, are largely flesh-eaters. This is especially true of the Anglo-Saxon race.

The same inference is suggested by the distribution of cancer in Scotland, where it is least frequent in the islands, where the population is comparatively poor, living largely on vegetable diet, than among the dwellers in richer districts and in towns, where more animal food is consumed.

This is, I think, all that we can learn from the larger geography of cancer.

It remains to consider how far its more special distribution within the bounds of a particular country throws light upon its causation.

In our own country we find cancer very unequally distributed, some parts showing a much greater prevalence of the disease than others; but it is extremely difficult to arrive at any general law on the subject.

It will be seen from the figures given by the Registrar-General that the mortality from cancer in England and Wales, as shown by the returns for registration counties, varies very much indeed in different parts of the country.

In 1898 the highest death-rates (and considerably higher than any other) were in Huntingdonshire and Rutlandshire, being 1414 per million, and 1261 respectively. After these, but with a considerable interval, come North Wales (1046), Shropshire (1028), and Westmoreland (1024).

The counties showing the lowest death-rate from this cause were in 1898: Durham, with cancer death rate, 568; Monmouth, 620; South Wales, 641.

These figures, however, require a great deal of modification before they can adequately express the actual relations of the prevalence of cancer in different districts. In a disease such as cancer, the mortality from which is mainly confined to persons over thirty-five, the prevalence will largely depend upon the proportion of persons over that age in the population. If there is a large element of elderly persons in the population the rate will appear unduly high; if there is a large proportion of children and young persons, a really considerable prevalence of deaths from cancer may give an unduly low

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rate. Now it is well known that the proportion of persons of different ages in the population—in technical terms, the age-distribution of the population—varies much in different places, and that the general death rate is much affected thereby. In order, therefore, to give the rates, as applying to comparable population, Dr. Tatham, in the supplement to the 55th Annual Report for England and Wales, has calculated the rates of cancer mortality for that portion of the population of each district over 35, as ascertained by the census of 1891. The result is to alter the "crude death-rate" for various counties very greatly. Most of the counties which give the highest crude death-rate are among those with the highest corrected death-rate, but the position of some of them is much altered, and the disproportion between different counties becomes much less.

It will be seen that after making this correction London is the highest on the list. But the number of deaths from cancer in London is so much influenced by the number of persons coming up to the great hospitals from different parts of the country, and dying in London, that this rate is certainly higher than would be the rate for actual inhabitants of London. I have not discovered any means of estimating the proportion of excess which would be due to this cause. The actual origin of hospital patients is difficult to discover, since they have often been a short time in London before entering a hospital, and give a London address. But when it is actually known that a person dying from cancer belongs to another part of the country, a correction is made in the Registrar-General's returns.

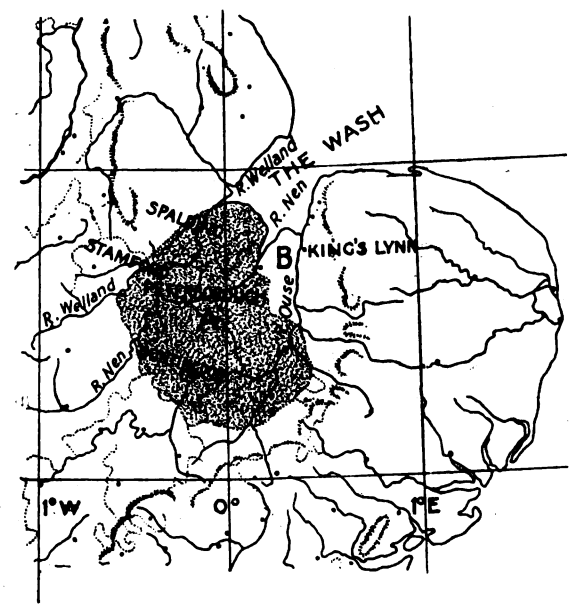
Apart from the case of London it is very difficult to discern any law which governs these variations.

We cannot say that agricultural or truly rural districts show any difference as compared with industrial or urban populations, for while three agricultural counties—Huntingdonshire, Cambridgeshire, Sussex—head the list, the three lowest are also counties to which the same description applies.

It is true that, with the exception of Sussex, a higher rate is observed in Northern and Midland counties than in the South and West; but here again Derbyshire, a Midland county, is an exception. We cannot say that lowland districts offer a constant difference as compared with hilly and mountainous districts; the great difference between North and South Wales being quite unexplained, though we may point out that South Wales has two large cities, Swansea and Cardiff, and has a large mining population, while North Wales has no large town.

Without going into further details, we may say that the differences between special districts point generally to something specific in the distribution of cancer, which is not accounted for by differences of latitude, of soil, or of the occupations of the inhabitants.

Dr. Tatham's report points out, however, one area of England where the cancer mortality is highest, which is thus described:



This area consists of the districts of Stamford, Bourne, Spalding, and Holbeach in Lincolnshire, Oundle and Peterborough in Northamptonshire, and the entire counties of Huntingdonshire and Cambridgeshire, with the exception of the registration districts of Caxton, Linton, and Newmarket. This area includes a population of over 300,000 persons.

Now, it may be said generally that this area is composed of flat and low-lying districts, and is mainly agricultural. It forms a basin, including old fen districts of Whittlesea Mere and the Bedford Level, and opening out into the Wash. I

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think the area might, perhaps, be extended to include some adjacent districts of Norfolk, namely, the districts of King's Lynn, Docking, and Walsingham, which also show a very high rate. This neighbourhood was at one time a very malarious district. Land drainage and other improvements have extirpated malaria, but not cancer, which has, as in other parts, greatly increased of late years.

This high death-rate from cancer seems to have no relation, positively or negatively, to the general death-rate from all diseases. For we find that while Huntingdonshire and Cambridgeshire have a general death-rate below the average, North Wales has an extremely high rate of mortality. Among the counties showing a low death-rate from cancer, Monmouth is below the average in general mortality; Cornwall is slightly above the average, and South Wales about the mean.

Nor can any direct or inverse relation be shown between the death-rate from cancer and that from phthisis.

On the whole, we must conclude that neither climate and soil, nor density of population nor occupation, accounts for these differences in the prevalence of cancer. Although the most distinctly cancerous area in England, just mentioned, is a low-lying, wet country, we cannot conclude that low-lying, damp soils are always distinguished by the prevalence of cancer; for in some such parts of England cancer is not very prevalent, and one of the cancer counties, North Wales, is a mountainous region. Just so, though malaria is found in marshy districts, there are marshes in many parts of the world; for instance, in Australia, where malaria is unknown.

In fact, the distribution of cancer, like that of malaria, seems to be governed by *specific* and not merely by general physical laws, though physical conditions may favour the prevalence of cancer, as they favour that of malaria. We know that in the case of malaria the specific law which determines its distribution is the presence of certain insects. There is nothing to forbid the supposition, though at present we have no clear evidence for it, that the prevalence of cancer in certain localities depends upon the presence there of some form of animal or vegetable life.

Another point strongly confirming the view that the distribution of cancer is specific, is that in areas of high death-rate from cancer, the prevalence is not uniform, but is made up of a number of smaller areas, some with a higher, some with a lower rate.

Further, these small areas can be again analysed into separate villages or parts of villages, with unequal rates; and even in these villages, it is certain streets, and in certain streets certain houses, which show the high rate of mortality.

Increase of Cancer.

This point has been illustrated by Dr. Tatham, whose authority confirms the belief that the increase shown by statistics is a real one, and not merely due, as has been suggested, to improvements in diagnosis and certification. I would only point out, in addition, that the increase in mortality from cancer in England is *progressive;* that is, the rate of increase is itself increasing. In 1871–80 the average annual rate of increase compared with the previous decade was 2·22 per cent.; the decade 1881—90, compared with the previous decade, was 2·45 per cent., and in 1891–99 had risen to 3 per cent. per annum.

Secondly, the rate of increase in cancer of the digestive organs is higher than the average for all organs, while the rate of increase in uterine and mammary cancer is below the general average. The increasing predominance of cancer of the alimentary tract is, from every point of view, most remarkable. In answer to the objection that improved diagnosis and certification accounts for the statistical increase in cancer, it is well to take the statistics of cancer in some organ as to which such errors can hardly arise; for instance, cancer of the tongue. The statistics from all sources show a striking increase of this form of cancer. The Reports of the Registrar-General for Ireland, in which for some years the numbers for special organs have been recorded, show a steady increase; the statistics for England and Wales show a still higher increase, amounting to over 5 per cent. per annum during the three years for which statistics of special organs are available, and hospital statistics confirm the result. Now, surely there is no room for mistake in the diagnosis and certification of a fatal case of cancer of the tongue. The statistics for cancer of the rectum, and cancer of the breast, of which the diagnosis in fatal cases is equally easy, show an undoubted though lower rate of increase.

On the causes of this increase it is difficult to

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pronounce an opinion. I agree with Mr. Roger Williams and Sir William Banks that the most plausible explanation *for this country* is the more abundant diet of the working classes and the increased consumption of meat. But we must remember, on the other hand, that cancer is increasing in other countries, and where the economical conditions are not the same. It is disheartening to see that material prosperity, sanitary improvements, decline of zymotic diseases and of tubercle, all those features of modern society which we are accustomed to boast of, certainly do nothing to diminish the prevalence of cancer, but possibly may tend to favour its spread.

FRACTURES OF THE RADIUS AND ULNA.

By W. ARBUTHNOT LANE, M.S.,
Surgeon to Guy's Hospital, and to the Hospital for Sick Children, etc.

BEFORE we consider the treatment of fractures of the ulna or radius, or of both bones, I would call your attention in the first instance to a few points in the mechanics of their movements upon each other and upon the humerus.

Being anxious to learn the latest teaching on the subject as provided for the student, I looked, up in the last edition of that most useful work 'Gray's Anatomy,' the descriptions of the actions of these several joints.

Under the head of actions of the elbow I found: "The portion of the joint between the ulna and humerus is a simple hinge-joint, and allows of movements of flexion and extension only;" and under the heading of actions of inferior radio-ulnar articulation: "It will thus be seen that in pronation and supination of the forearm and hand the radius describes a segment of a cone, the axis of which extends from the centre of the head of the radius to the middle of the head of the ulna. In this movement, however, the ulna is not quite stationary, but rotates a little in the opposite direction. So that it also describes the segment of a cone, though of smaller size than that described by the radius. The movement which causes this alteration in the position of the head of the ulna takes place principally at the shoulder-joint by a rotation of the humerus, but possibly also to a slight extent at the elbow-joint."

I think I will be able to show you that these statements are very misleading, as is usually the case in the text-book descriptions of the physiology of the skeleton. If you examine your own arm carefully you will see that there are two distinct movements of the radius of the ulna. One is the movement of pronation and supination as described by the anatomist. In this the ulna is fixed upon the humerus while the radius moves upon it, so that its styloid process describes a portion of a circle around the styloid process of the ulna, while its head rotates upon the radial head of the humerus and upon the lower sigmoid cavity of the ulna. In this movement the ulna is fixed upon the humerus, and the styloid process does not move laterally. The other movement is a much more complicated one, and is perfectly different in every detail. It is the mutual movement of the bones on one another and on the humerus, which takes place when, with the pronation and supination, force is exerted upwards along the length of the radius, as in the movement of driving in a screw. In this there is no movement whatever in the shoulder-joint so long as the range of pronation and supination is not excessive.

The *radius rotates on its own axis*, the styloid process moving around a point which may roughly be regarded as the centre of its carpal facet. Most of the force is transmitted upwards through the rotating radius to the radial head of the humerus directly and in smaller proportion through the interosseous membrane and ulna to the humerus indirectly. In order that the radius shall rotate accurately and securely on its longitudinal axis, the ulna moves around a transverse axis gradually varying in its obliquity upon the humerus, first in the direction of extension, and then, having reached a limit of extension, it moves in the direction of flexion upon the humerus, but through a much smaller angle. Associated with these movements of extension and flexion of the humero-ulnar joint there is one of abduction of the ulna upon the humerus. In consequence of these combined movements the lower end of the ulna describes portion of an arc of a circle, whose centre corresponds to the axis of the radius around which it rotates. There is no rotation whatever of the ulna around its own axis.

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These movements, then, are perfectly distinct. In the first the ulna is fixed, and the radius rotates around it; in the second the radius rotates on its own axis, and the ulna moves around it, the form of both bones being evolved to meet their capacity to bear these mechanical relationships to one another. In neither case does any movement in the shoulder-joint occur, except when the movements are excessive, or when the combined movement is performed with a completely extended arm.

The second movement is most important mechanically to the individual, and in order that it be performed normally it is of vital importance that the ulna as well as the radius be normal in form. The surgeon, being unaware of the important part played by the ulna in the movement, and being imbued merely with the scanty information of a limited and incorrect nature as to the physiology or use of the part as supplied by the text-books on anatomy, regards fractures of the ulna as of little importance as compared to those of the radius. This a little experience will show you to be very far from true. In our general hospitals fractures of the upper extremity, and especially those of the forearm, are regarded as of such little interest and so unimportant that they are relegated to the care of the least experienced members of the resident staff, and are treated with splints of the roughest description possible and without any scientific basis whatever. Consequently the mechanical disability resulting from this treatment is very great, and the results, which are disastrous mechanically, abound. Yet no one pays much attention to these failures, but everyone is prepared to be unconscious of their existence, and to make the most obviously ridiculous false statements in regard to the success they have obtained in their treatment of fractures of these bones without the slightest hesitation. My experience in this direction has led me to wonder if surgeons are equally inaccurate in their statements about other conditions. I trust sincerely this is not the case, but I have certainly grown very much less credulous than I used to be, and now always prefer to see any assertions amply verified by facts.

The mechanical conditions in the forearm are very different from those in the leg. In the latter the bones move on one another very slightly indeed, and any alteration in the axis of the fragments produces disability by altering the direction of the lines of pressure through the several joints which are affected. Even ankylosis of the tibio-fibular joints or of the tibia with the fibula of itself results in no considerable disability. Again, in the leg the form of the fracture is usually spiral, and the overlapping of the fragments brought about by the driving of the chisel-shaped fragments upwards and downwards in diverging directions into the surrounding soft parts is considerable, and the accurate coaptation of the broken surfaces is correspondingly difficult, even when the seat of fracture is freely exposed.

In the case of the forearm the movement of the bones on one another is a matter of vital importance to the perfect performance of their functions, and any alteration in the axes of the fragments to one another is followed by a corresponding amount of physical depreciation. The bones are broken more or less transversely, a spiral fracture being very rare, owing to the mechanical conditions present. Consequently there is not the same overlapping of fragments as in the leg, nor the same tendency for them to diverge from a common axis, as the ends are more or less flattened, and not chisel-shaped. The difficulty of bringing the fragments into apposition is very much less in the arm than in the leg; but even if this is effected, it is often impossible by manipulation and splints to render and retain the axes continuous and avoid any angularity. On the other hand, these fractures of the radius and ulna lend themselves much more readily to operative interference, as the accurate coaptation and fixation of the fragments is comparatively easy once they are freely exposed.

If, however, union has taken place in such a manner that the fragments form angles with one another, very considerable disability may result, and the operative measures necessary to restore the bone or bones to their normal form are often very difficult to carry out, owing to the necessity of cutting them in four distinct planes if both are at fault. In my experience these cases of considerable mechanical disability following fracture of the radius or ulna, or of both bones, are very common, and I have operated on a considerable number. I have frequently been struck by the fact that a very small angle between the fragments results frequently in a disability which is, at first

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sight, quite out of proportion to it. This is particularly the case with fractures of the ulna. Again, with or without the existence of an angle between the axes of the fragments, there is not uncommonly present a varying degree of rotation of the fragments in the longitudinal axis of the bone.

I will confine myself to giving one typical instance of this class of fracture, as they are all very much the same in their general characters. When they occur in young people an attempt is made to remove as much of the angle between the fragments as possible by the abundant deposit of new bone about both fragments along their entire length, in obedience to the demand made by the transmission of force in new directions, and out of this new material, and including more or less of the old shaft, a new one is formed. The younger the subject the more perfect is the development. After a certain period in life any improvement brought about in this way is small.

A. B—, æt. 18 years, a driver in the Royal Horse Artillery, was thrown off a limber on November 6th, 1901, and sustained a fracture of both bones of the forearm. He was treated by means of an internal angular splint, the forearm being fixed in the semi-prone position.

He was invalided out of the service on May 13th, 1902, as the condition of his arm rendered him unfit to perform the duties of a soldier.

He came under my care on May 26th, 1902. The forearm was retained in the semi-prone position, and it was possible to rotate the radius on the ulna in the direction and in the manner described by the anatomist as pronation through an angle of about 45°. Any attempt at the mutual movement of the bones on one another, such as occurs in rotating the radius on its own axis, was of no avail. Financially his disability is complete as far as soldiering is concerned, and this has resulted solely from the stupid conservatism of surgery, if in the present day it is fair to apply the term surgery to such a gross incapacity to understand the simplest mechanics of the skeleton.

This patient's shoulder-joint was perfectly normal, and it was clear that in him the inability of the ulna and radius to move on one another was due solely to the alteration in the form of these bones and not to any incapacity to rotate the humerus around its own axis. I mention this, as it has been asserted that the humerus rotates considerably in pronation of the forearm. I have not been able to satisfy myself that any appreciable movement takes place in the shoulder-joint, except when the elbow-joint is fully extended, other than that necessary to secure the bone firmly in position when a greater proportion of the transmitted force is exerted upon the radial head than upon the trochlea tending to rotate the humerus. Therefore, in order to permit of force being exerted along the radius rotating on its own axis in extreme extension of the elbow-joint, the shoulder has to be raised so that the humerus and ulna locked together in extreme extension can extend themselves upon the radius which alone remains fixed. Any movement of the humerus on its own axis comes about so that force may be transmitted to the shoulder girdle at the greatest mechanical advantage and not to allow of the movement of the ulna on the radius, which I believe takes place solely at the elbow-joint.

The mode in which I operate on these bones with a view of dividing them and restoring them to their normal form is as follows :—The fractures are exposed in the freest manner possible, the bone being denuded of its soft parts for some distance about them. The upper fragment is accurately defined, and is cut through obliquely very close to its connection with the lower fragment. The forearm is then placed in a position of complete supination, when the relationship of the lower fragments to the upper can be carefully gauged and the directions of the planes in which they must be divided accurately determined. The sawn surfaces are then fitted together, and are retained immovably in apposition by silver wires, which perforate them, and, if necessary, by additional encircling loops. I would again call attention to the importance of using only virgin silver wire, and of heating it to a red heat before using it, in order to render it as flexible as possible. Before the wounds are closed the surgeon should make sure that the forearm is capable of performing perfectly the several movements required of it.

AMONGST the books which will shortly be published by the firm of J. and A. Churchill are—The seventh edition of 'The Diseases of Children,' by Drs. Goodhart and Still; the second edition of 'A Manual of Bacteriology,' by Dr. Hewlett, Professor of Pathology and Bacteriology in King's College; and the fourth edition of Volume II of Fagge's 'Text-book of Medicine,' under the editorship, as hitherto, of Dr. Pye-Smith, F.R.S.

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WITH MR. TREACHER COLLINS

AT THE

MEDICAL GRADUATES' COLLEGE AND POLYCLINIC.

MARCH 21st, 1902.

Tubercle of the Ocular Conjunctiva.

THE first case I have to show you, gentlemen, is one, the diagnosis of which from its clinical characters alone is very difficult, but it is made definite by the specimen I have put under the microscope.

The man is æt. 67, and came first to the hospital on February 27th, 1902, complaining of inflammation of his left eye, which he had noticed six or seven weeks. The ocular conjunctiva was injected, and upwards and inwards a short distance from the corneal margin was a raised, nodulated, pinkish-grey patch, half an inch long and about a quarter of an inch in its broadest part. There was no ulceration of the surface, and no enlargement of the pre-auricular gland.

No specific history could be elicited. He had had no injury. When he was twelve years of age he had a white swelling on the right knee. Two years ago he had his right leg amputated through the thigh after an accident.

The nature of the growth seemed uncertain; in a man of his age a growth of the ocular conjunctiva would most likely be of a malignant character, either a sarcoma or an epithelioma. I determined to cut out a small piece for microscopical examination, and as you will see by the sections, it shows the typical histological appearance of tubercle. It has the typical giant-cell systems. After the histological examination there was not sufficient of the specimen left to examine for bacilli.

The position of the growth on the ocular conjunctiva is rather an unusual one for tubercle. The commonest way for tubercle to attack the conjunctiva is in the form of an ulcer on the palpebral portion of that membrane. Before the microscopical examination had been made, the patient was treated with iodide of potassium, and under its influence the growth has certainly diminished in size, but now, having ascertained its nature, I propose to excise the whole piece of the conjunctiva which is involved. The case is a good example of the value of microscopical examination where the diagnosis is in doubt. The prognosis is, of course, very good, for after excision the growth ought not to recur locally. It is, however, doubtful whether or not it is a primary lesion.

Rupture of the Sclerotic from an Injury with a Cow's Horn.

If you examine an antero-posterior section of the eye you will find that the thickest portion of the sclerotic is at the posterior part around the optic nerve. It gradually tapers in passing forwards to the equator where it is thinnest. A little past the equator it receives an accession of fibres from the recti muscles, and then again becomes slightly thinner on approaching the corneal margin. At the equator, where the sclerotic is thinnest, it is supported by the tendons of the recti and oblique muscles externally, and the retina and choroid internally. The thinnest portion of the sclerotic, which is not so supported, is a short distance from the sclero-corneal margin, about three millimètres external to it. It is in that position where we meet with a rupture of the sclerotic when the tension of the eye is suddenly increased by external pressure. The rupture extends into the extreme periphery of the anterior chamber, and the vitreous chamber does not become opened up unless the suspensory ligament is also ruptured and the lens displaced.

A rupture of the sclerotic occurs always on the opposite side to that on which a blow is inflicted, and as the eye is protected to some extent above by the brow, and on the inner side by the nose, the most frequent seats of rupture are either directly upwards or upwards and inwards, blows being usually received from either below or the outer side.

The case I have now to show is an exception to this rule; the rupture is situated in the upper and outer part, and the man received a blow on the lower and inner part from a cow's horn. There is a small foreign body beneath the ocular conjunctiva marking the position at which the blow was inflicted. An injury of the eye by a cow's horn is apparently not a very infrequent accident amongst those who are engaged in agricultural pursuits. Fuchs, in his text-book on ophthalmology, describes how a man received an injury to each eye from a cow's horn at an interval of two

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or three years. In each eye there was a rupture of the sclerotic and an escape of the lens through the rupture, but the structures at the back of the eye were very slightly, or not at all, injured, and the man afterwards was able, with cataract glasses, to see to read fairly satisfactorily ; so that in this case the man had had an extraction of both lenses performed by a cow with better results than sometimes attend the operations of a surgeon.

I was speaking once to an American ophthalmic surgeon of these cases of injuries to the eye by cows' horns, and mentioning this case described by Fuchs, and he said : "Yes, I have seen a man who had a rupture of the eye by a cow's horn, and this man had a rupture of his other eye a few years later by a blow with the tail of the same cow." This patient here is a man æt. 45, and he received the injury sixteen days ago. I saw him the following day ; the conjunctiva was swollen and œdematous, and there was also much blood beneath it. The line of rupture in the sclerotic could only indistinctly be made out on account of the blood, but the conjunctiva appeared to be intact over it. There was a large quantity of blood in the anterior chamber, and it was difficult to determine whether or not there was any displacement of parts in the interior of the eye.

Some horizontal striæ could be seen in the upper part of the cornea, evidently due to rucking of Descemet's membrane. Vision equalled only hand movement, and the tension was — 2.

Now, to a great extent, as you see, the blood has become absorbed. The line of rupture can be distinctly made out as a grey-coloured line up and out, three millimètres from the sclero-corneal margin, and the iris in the position of the rupture appears to be absent. It looks just as though an iridectomy had been performed in that direction. Really the iris has become incarcerated in the gap formed in the sclerotic. There is still blood-stained membrane filling the pupil, and only a dull reflex can be obtained from the fundus. His vision is now equal to counting fingers at four feet, and the tension is normal.

As there is a prospect of the man ultimately obtaining useful vision, and as no actual perforation into the eye from without has occurred, the conjunctiva having always remained intact, I do not propose to remove the eye, but shall keep a careful watch on the patient for some time, for fear that sympathetic trouble may ensue. A few cases have been recorded of rupture of the globe in which sympathetic inflammation of the fellow eye has followed, and some even where there was no apparent perforation of the conjunctiva. Probably in such cases a conjunctivitis had occurred, and the inflammation had spread to the uveal tract, along a portion of it which had prolapsed through the rupture, and lay in contact with the conjunctiva.

Congenital Defective Movement of one Eye making it closely simulate an Artificial Eye.

I suppose that all of us at one time or another in our student days have been taken in by an artificial eye, imagining it to be a real eye. This patient presents the opposite condition ; that is to say, she possesses a real eye which closely simulates an artificial one. Many excellent observers to whom I have shown her and asked their opinion of the case, have at once smiled and said, "Oh yes, artificial eye, of course." Many acquaintances have asked her whether she had not got a glass eye.

I will just point out to you the many points of resemblance which this patient's left eye presents to an artificial one.

She is a woman æt. 30, and she states that her left eye has been defective as long as she can remember. It was operated on because it turned in eleven years ago. You will observe that there is a partial ptosis, and a slight sinking of the eye in the orbit, so-called enophthalmos. Now, an artificial eye generally looks less prominent than a normal one, and there is almost always also slight drooping of the upper lid over it.

This patient has limitation of movement of this eye upwards and outwards ; on looking inwards there is some rotation downwards. A limitation of movement is, of course, one of the defects which an artificial eye presents.

There is also in this patient extreme sinking of the caruncle and some retraction of the inner part of the lower lid, which greatly intensifies the resemblance which this eye presents to an artificial one.

The limitations of movement in this patient's eye, I think, are congenital, due to some faulty insertion of the muscles into the globe ; at any rate, the history seems to suggest that. The extreme sinking of the caruncle and the retraction of the

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lid are, however, I think, the results of the operation which she says was performed upon it, and which was probably a tenotomy of the internal rectus.

The patient has very defective sight in the eye, only being able to distinguish light from dark with it. The optic disc is pale, the nerve apparently being ill-developed.

The pupil is active to light, which feature should at once be looked for to distinguish it from an artificial eye, for as yet we have not reached that acme of perfection by which we can get an artificial eye with a pupil active to light.

Results of a Plastic Operation for Ectropion.

This patient when I first saw him had extreme ectropion of each lower lid. Their margins had become drawn down some distance past the lower border of the orbits nearly down to the alæ of the nose, the condition not only being very unsightly but very troublesome to the patient on account of the epiphora it gave rise to. The cause of the ectropion was cicatrisation following lupus, the amount of which is very extensive.

When I first saw him there was still some inflammatory infiltration about the skin, and he was treated for a time at the London Hospital with the X rays, which resulted in the great improvement in the condition of the scar tissue. I showed him here on a previous occasion, shortly after I had performed a plastic operation on the left side; since then I have performed a similar operation on the right side.

It was impossible, on account of the extensive cicatrisation, to twist any piece of skin, leaving a pedicle attached, from some neighbouring part in order to make a fresh lower lid, nor was it possible to perform any of the numerous and ingenious gliding operations which have been invented. I determined, therefore, to transplant a piece of the whole thickness of the skin from the upper arm to the lower lid, and this is the operation which I have performed on each side, and as you see with a very satisfactory result, there now being only a slight amount of ectropion at the outer angle of each lid.

The procedure of the operations was as follows:—I first made an incision along the margin of the lower lid about five milliampères distant from it, and dissected up the border of the lid so as to allow it to come into its normal position. I then stitched the margin of the two lids together with horse-hair sutures, and there was a large raw surface left which it was necessary to fill up.

I cut a paper pattern of the size and shape of this raw area, and marked out on the man's upper arm a piece corresponding in shape but somewhat larger. Where you see the cicatrix has been left I dissected up this piece of skin, as far as possible free from any subcutaneous tissue. Directly the skin was separated it shrank up considerably, and so became almost exactly the size which was required. I then laid it on the raw surface which had been left beneath the margin of the lower lid, and secured it in that position, not by passing sutures through the transplanted flap, but by making a network of sutures overlying it which were anchored to the skin above and below by passing them through a small fold in the way suggested by Dr. Argyll Robertson. I dusted on iodoform and applied a dry dressing, which was left on for ten days. On the removal of the dressing the whole of the flap was found to have taken, there being no sloughing whatever, and for some weeks after the operation the margin of the lid was in perfect position, but since that time the flap has somewhat contracted, and, as you see, there is still a slight amount of eversion at the outer angle.

You can judge how much the transplanted skin has shrunken by seeing the size of the cicatrix on the upper arm in the position from which it was taken, and comparing it with the transplanted piece of skin, the outlines of which are still clearly discernible.

In order to get large areas of transplanted skin to take like this, I attach a great deal of importance to two things:—(1) The flap should not be in any way injured by the passing of sutures through it, because, as I have several times seen, the places where such sutures are inserted become the starting points from which sloughing subsequently ensues; (2) The application of a dry dressing; a moist dressing seemingly favours sloughing.

It is further interesting to note that the piece of transplanted skin on the right side has a number of black hairs growing from it, which look quite unnatural in connection with the lid, which normally is entirely devoid of hairs.

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A CLINICAL LECTURE

ON

ACUTE PLEURISY WITH EFFUSION.

Delivered at the Hospital for Consumption and Diseases of the Chest, Brompton.

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PLEURISY with effusion is a disease about the nature of which our views have changed a good deal during the last fifteen years or so, and there are certain points concerning the prognosis and treatment connected with our present knowledge, which I wish to bring prominently before your notice as of great practical importance. While formerly so-called acute idiopathic pleurisy was thought to be an obscure inflammatory affection which was generally set up by chill or cold, lately it has been recognised that more commonly than not it is nothing more or less than tuberculous inflammation of the pleura. Weighty evidence may be brought forward in favour of that view; a view which was advanced first by physicians of the French school, some of whom go so far as to maintain that pleurisy *a frigore* does not exist. Now, the evidence which we have as to the nature of acute idiopathic pleurisy comes from several different and independent sources. It is derived from the experience of the post-mortem room, from the results of examination of the fluid obtained during life by aspiration from the pleural cavity and of inoculating animals with this fluid, from the results of testing persons by means of tuberculin during their convalescence after pleurisy, from the after-history of persons who have recovered from an attack of pleurisy, and from the frequency with which we find a history of pleurisy previous to the outbreak of pulmonary tuberculosis.

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The evidence which one obtains from post-mortem examinations is perhaps the least conclusive, for idiopathic pleurisy generally terminates in recovery, and only a small proportion of the cases pass into the post-mortem room. For instance, during the last fourteen years over six hundred cases of pleurisy were admitted into St. Thomas's Hospital, and of these only fourteen terminated fatally. Four of these fatal cases were found to be distinctly tuberculous. Of the others, three were complicated with pneumonia, one with pericarditis, and one was secondary to cardiac disease, while the nature of the remainder is not stated. I think we may safely conclude that more than four were tuberculous, for it is often very difficult to recognise tubercles in inflammation of the serous membranes without a minute microscopical examination and special staining, which was not carried out in every case. Some years ago Professor Osler* carefully analysed the post-mortem records of 101 cases of pleurisy, and thirty-two of these were ascertained to be definitely tuberculous, although these 101 cases included many in which pleurisy complicated other affections. The evidence of the post-mortem room, then, such as it is, is not incompatible with the contention that a fairly large proportion of cases of pleurisy, whether idiopathic or not, are of tuberculous origin.

The next link in the chain of evidence is the result of examination of the serous effusion. In the majority of cases the serous effusion is found to be perfectly sterile, and the most careful microscopic examination does not reveal the presence of any micro-organisms, and attempts to obtain cultures from it have signally failed. You may, perhaps, ask how can this justify a belief that the disease is due to micro-organisms. But although the fluid is sterile the effects of the inoculation of guinea-pigs with the fluid show that in a large proportion of the cases tubercle bacilli are really present, although that cannot be demonstrated by microscopical examination or cultivation experiments. Inoculation of guinea-pigs does not invariably prove successful, even when performed with fluid from cases undoubtedly tuberculous. So that while a positive result proves the tuberculous nature of the pleurisy, a negative result does not establish the contrary. And we shall not be wrong in concluding that even a larger proportion of the cases is tuberculous than is shown by the now well-known inoculation experiments which have been carried out by French and German scientists.

Chauffard and Gombault* injected into the peritoneal cavities of guinea-pigs 3 c.c. of serum from twenty cases of pleurisy, and in ten cases out of twenty they succeeded in producing tuberculosis. A German investigator, Aschoff,† produced even more striking results. He injected 3 c.c. of the serum into the peritoneal cavity, and out of twenty-five cases where the pleurisy was either certainly or very probably tuberculous he got a positive result in seventeen or 68 per cent., and a negative result in eight or 32 per cent. Out of twelve so-called idiopathic cases, cases which clinically could not be differentiated from the ordinary pleurisy *a frigore*, only three gave a negative result; that is to say, in only 25 per cent. of the cases did he get a negative result. The experiments of another observer, Eichhorst,‡ go to show that the larger the amount of serum which is used for injection, the smaller is the proportion of failures. When he injected 1 c.c. of fluid, in ten out of eleven cases the animals remained healthy. In the remaining case, tuberculous infection of the lymphatic glands was produced. In later experiments he used a larger quantity, 15 c.c., and when this was done, no fewer than fifteen out of twenty-three guinea-pigs were infected, that is to say 65 per cent.; in other words, two thirds of the cases of serous pleurisy proved to be tuberculous. Hence we may conclude that probably about four out of five cases of idiopathic pleurisy with effusion which are ordinarily seen in practice are tuberculous.

The next source of evidence is the result of injection by means of tuberculin. Although testing patients with tuberculin has gone so much out of fashion, if not actually into discredit, I consider that it is in certain cases extremely valuable, and if properly used free from danger. I am of opinion that if we do not use it, we fail to avail ourselves of a very important means of diagnosis, one which may be of much advantage to our patients. It is of the greatest moment to attain certainty as to whether a person is tuberculous or not, for if he be

* 'Shattuck Lecture,' Boston, 1893.

* 'Bull. Soc. méd. des hôp. de Paris,' 1884.

† 'Zeitschr. f. klin. Med.,' Bd. xxvi.

‡ 'Corr.-Bl. f. Schweiz. Aerzte,' July, 1895.

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found to be tuberculous he should be acquainted with the fact and should be treated accordingly, while if he be not tuberculous the same precautions need not be taken and he may be allowed to resume his ordinary mode of life at an earlier period. One result of the disfavour, or discredit, into which tuberculin fell soon after its introduction is that the evidence derived from this source as to the tuberculous character of pleurisy is not very large. But in the early days when tuberculin was widely used, of fifteen cases tested thirteen were found to react. As it is very exceptional for a person who is not tuberculous to give a positive reaction to tuberculin, that is strong evidence. The tuberculin test is of course not infallible, and sometimes a reaction occurs in non-tuberculous diseases, as in a case mentioned by Osler,* where a patient with pleurisy was tested with tuberculin and reacted, and where afterwards it was found that the disease was cancer and not tubercle.

I have not used tuberculin in cases of pleurisy to any great extent, principally because I have reserved it for the cases in which I have doubted the presence of tubercle, and these have formed only a small proportion of the cases which have come under my care. When I have employed the tuberculin test I have had the satisfaction of having my opinion confirmed by a negative result. The method of testing I employ is that recommended by Professor Koch himself. It is first ascertained that the temperature is maintained at a normal level. I use a recently prepared 1 per cent. dilution of Koch's old tuberculin, and to begin with inject ·1 c.c. of the dilution deeply into the subcutaneous tissue of the back or buttock either early in the morning or late at night. If only a slight rise of temperature occur, such as half a degree, then in two days another injection of the same amount is repeated. The reason the same dose is given is that you sometimes get a marked reaction on repeating it, and it is important to avoid any excessive reaction. If no rise occur the first time, or only a slight rise when the same dose has been repeated a second time, then one injects ·2 c.c. If there is still no reaction, or only a slight one after two injections of ·2 c.c., then one proceeds exactly as before, but injecting ·3 c.c. If, finally, no reaction occur with ·5 c.c., then one is justified in concluding that the case is not tuberculous.

But perhaps the most valuable evidence of all is derived from the after-history of patients who have suffered from pleurisy with effusion. If you inquire into the matter you will be very much struck by the large number of phthisical patients who give a history of an attack of pleurisy before the onset of any symptoms of phthisis, and often it is known that the pleurisy was accompanied by effusion. And if you follow up your cases of pleurisy with effusion, as those of you who are in general practice have opportunities of doing much more than we at hospitals, you will be impressed with the large proportion in which phthisis later on appears. In the year 1890 Dr. Barrs,* of Leeds, traced the subsequent history of fifty-seven patients who had been treated for serous pleurisy in the Leeds Infirmary between the years 1880 and 1884. Of these fifty-seven patients he found that no fewer than thirty-two were dead, and that eighteen out of the thirty-two had died of tuberculosis, fourteen from phthisis, and four from other tuberculous affections. Professor von Ziemssen has recorded his opinion that of adults who have had an attack of pleurisy, nearly one half ultimately died of tubercle. Dr. Bowditch,† of Boston, U.S.A., brought forward some years ago some statistics chiefly drawn from cases in private practice, and embracing an experience extending over a large number of years. Out of forty-nine cases of pleurisy which had been seen between the years 1849 and 1869 no fewer than twenty-one died from phthisis, and only eighteen were still alive and well in 1889. Of forty-one seen between the years 1869 and 1879 nine were dead from phthisis, one was living and phthisical, and twenty-nine were alive and well. If the investigation were made now it would probably be found that an additional number had shown themselves tuberculous. Dr. Bowditch's cases might have been more striking had he separated, as he did not do, the cases of empyema and dry pleurisy from the cases of pleurisy with effusion, for the after-history of cases of empyema is much more favourable than that of pleurisy with effusion. Ricochon‡ found

* Op. cit.

* 'Brit. Med. Journ.,' 1890, vol. i, p. 1059.

† 'Medical News,' Phila., 1889, vol. lv, p. 63.

‡ Verneuil's 'Etudes experimen. sur la tuberculose,' 1887, vol. ii.

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that out of thirty-three cases of pleurisy fourteen died obviously tuberculous, and seven others showed signs of tubercle either before or after the attack of pleurisy. Fiedler,* of Dresden, investigated 112 cases which had been treated for serous pleurisy by him in Dresden, and of these, twenty-five died of tuberculosis, sixty-six became certainly or probably tuberculous, and only twenty-one out of the 112 were in good health one or two years after the attack of pleurisy. I have not had the opportunity of following up a sufficient number of hospital cases to be able to bring forward similar figures from my own experience. Anyone who has done this kind of investigation in the case of hospital patients must know that it involves much time and labour, especially in London, where the difficulty of tracing patients after a lapse of years is very great. But of the cases of pleurisy with effusion which have occurred among private patients or students, I can remember many instances where phthisis has supervened later on; and I do not think, after all the statistical work that has been done in so many different quarters, that further investigation is necessary. Taking into account all the different kinds of evidence I have mentioned, I consider that there is enough testimony to show that acute idiopathic pleurisy is, in a large proportion of cases, a tuberculous disease.

I must now pass over very briefly the symptoms and physical signs of idiopathic pleurisy with effusion. The ordinary symptoms are pain, pyrexia, cough, dyspnœa, quickened pulse, and constitutional disturbance. The pain of pleurisy is seldom so severe as that of acute pneumonia at the beginning, but it is decidedly more persistent. It may be very intense in cases with slight signs. It is characteristic that it is aggravated by taking a deep breath or by coughing. The pain is generally accompanied by local tenderness on pressure over the chest wall. By the time the effusion has developed to an appreciable extent the pain usually has subsided. Sometimes pain is absent throughout the whole of the illness. The course of the temperature in pleurisy with effusion is somewhat variable. Pyrexia usually lasts for two or three weeks, but as a rule the temperature is not very high, ranging between 100° and 102° F. In some cases the pyrexia is of quite short duration, lasting only a few days, while in other cases the pyrexia may continue for one or two months. Cough is sometimes a very troublesome symptom at the beginning of the attack, being no doubt reflex from the irritation of the costal pleura. It is often excited by a deep breath, especially when that gives rise to pain, and is generally not merely dry and useless, but irritating and worrying. Sometimes there is a little mucous expectoration, and sometimes there is accompanying bronchial catarrh. Dyspnœa in pleurisy with effusion is seldom a marked objective symptom until the effusion becomes a large one. There is then a considerable degree of dyspnœa on exertion, although there may be little or none while the patient keeps quiet. Usually the breathing is shallow when pain is at all a marked symptom, and while the rate of respiration is somewhat quickened there is not the altered pulse-respiration ratio which one observes in acute pneumonia. The pulse is moderately rapid, weak, and of low tension. There is sometimes sweating at night, as in pulmonary tuberculosis, especially if the bedroom be hot and the bedclothes too abundant, but constant or profuse sweating is uncommon, and generally points to the presence of a purulent effusion. The urine is usually concentrated and diminished in amount, while there is pyrexia and increasing effusion, and returns to a normal condition as the effusion subsides. Constitutional disturbance is shown by malaise and general weakness.

As regards the course of the disease, as a rule it is towards recovery. But the illness is somewhat tedious, and it is usually from four to six or eight weeks before the patient is able to be up and about again.

The physical signs of pleurisy with effusion are so well known that there is but little that one need say about them. Everyone is familiar with the points of distinction between the physical signs of pleural effusion and those of consolidation, but still even the most experienced physicians will sometimes be in doubt as to whether they are dealing with fluid or with a solid lung. The difficulty does not usually arise with large effusions, as you practically never have a large effusion without getting displacement of the neighbouring organs and of the diaphragm and mediastinum. If you have a large effusion of the left side you find the

* 'Volkmann's Sammlung,' 1882.

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mediastinum and the heart displaced to the right; if you have a large effusion of the right side you find the mediastinum and the heart displaced to the left. As the result of the descent of the diaphragm the liver is depressed on the right side, and Traube's semilunar space is diminished or obliterated on the left side. It is in the case of small or moderate effusions that one may be in perplexity. The presence or absence of vocal fremitus is a very important aid to diagnosis. If you get a distinct and decided vocal fremitus over a dull area you may be practically certain that there is no fluid. The opposite, however, does not always hold good. Over a consolidated lung you may sometimes get a decidedly enfeebled fremitus. I find that students are very apt to be misled by the character of the breath sounds. Bronchial or tubular breathing is not uncommonly present over a pleural effusion. Usually the bronchial breath sounds have a distant quality, but sometimes they are as distinct as over a consolidated lung. Suspect fluid if with bronchial breathing you get diminished fremitus. A good deal of attention has been paid by some French physicians to the varying areas of dulness with changes of posture of the patient. The original idea of Piorry that the effused fluid in the pleural cavity moved about as freely as the liquid in a water-bottle has been long known to be incorrect. Free movement of the fluid in the pleural cavity with change in posture only occurs when air as well as liquid is present in the cavity. When the effusion is of small amount, the liquid tends to occupy the most dependent parts of the thorax, and when the patient is examined in the sitting posture dulness may be observed only in the lateral aspect of the chest. In effusions of moderate amount, the patient being seated, the upper limit of dulness in front and laterally is a horizontal line. Posteriorly at a distance of some two or three inches from the vertebral column the line of dulness abruptly falls, forming a triangle in which resonance persists below the general level of dulness. It is useful to know that in some cases of moderate effusion a change in the form of dulness with a change in the position of the patient may be observed. While in the sitting posture the anterior level of dulness is horizontal, when the patient lies down it tends to assume a somewhat slanting direction, sloping downwards and inwards. Such a change of level of dulness when present points to fluid. When there are large effusions there is no alteration in the shape of the dull area with the change in posture of the patient. Occasionally an effusion spreads itself out between an indurated lung and the thoracic wall, leading you perhaps to imagine from the extent of the dulness that you are dealing with quite a large effusion of fluid. Sometimes we find that an effusion is suspended in the upper part of the thorax as the result of adhesions in the lower part, or the upper anterior border slopes downwards and inwards in consequence of adhesions over the upper part of the lung.

One is often asked whether it is possible to distinguish a serous from a purulent effusion. It is certainly seldom that we can do so by means of physical signs. Some years ago a good deal of attention was drawn to a sign called after the distinguished Italian physician who described it: Baccelli's sign. It was held that where the effusion was serous there was whispering pectoriloquy at the upper level of fluid, while this was not present when the effusion was purulent. I simply mention this to impress on you that no reliance whatever can be placed on it. You may get whispering pectoriloquy in cases of empyema, and you may not get it in serous effusion. You usually find leucocytosis in empyema, and this is less commonly present in serous effusions. Pyrexia occurs in both, and is usually more persistent in purulent than in serous effusions, but in both it may be absent. When you get œdema over the dull area that points to a purulent effusion. But what I attach most importance to, in coming to a conclusion as to the nature of an effusion, is the history of the onset and mode of progress of the illness. Most of the cases of empyema which we see in adults follow acute pneumonia; and if you find that there is a history of acute and sudden onset, the patient having become seriously ill within a few days, you may be pretty certain that the effusion is purulent. If the illness has come on in a more gradual and insidious way, the symptoms during the first week or so perhaps not being so severe as to keep the patient in bed, the probability is that you are dealing with a serous effusion. In the case of children, however, you must bear in mind that purulent effusions are, on the whole, more common than serous; and when pleurisy follows an attack of one of the specific fevers, such as

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measles or scarlet fever in a child, then there is a strong probability that the effusion is purulent. Whenever you are in doubt in the matter, of course what you ought to do is to make an exploratory puncture. But even when you have drawn off fluid by an exploratory puncture it does not make you absolutely certain as to the nature of the case. I remember a case which I saw with my friend, Dr. Macevoy, of Brondesbury, where there were signs of fluid, and on tapping posteriorly we got pus, and a small empyema at the base of the left lung was surgically dealt with in the usual way. The opening soon, and as it appeared prematurely, closed up, for the patient continued to have pyrexia and kept losing ground. As there were still signs of fluid we aspirated again more anteriorly, and this time drew off a pint of clear, not purulent fluid. The pyrexia continued, and when I next examined I found signs of pneumothorax over the front of the left lung. I felt little doubt that we had to deal with a case of tuberculosis of the lung and pleura. The patient gradually got worse, and grew weaker and more and more wasted. But one day she had a violent fit of coughing and expectorated a large quantity of pus, and from that time improvement set in. Eventually she got quite well, and she has remained perfectly well ever since. This clearly was a case of loculated empyema behind, with a serous effusion in front.

A disease which you might not think of, as we do not often meet with it in this country, namely, abscess of the liver, sometimes simulates pleurisy, and I have recently had a case of this kind under my care at St. Thomas's Hospital. The patient was a young man who had been in the South African Campaign in the year 1900. He had had dysentery in Africa for which he was invalided home, but he arrived in England perfectly well, and was able to take a situation as footman, remaining in good health for about a year. About the month of September, 1901, he was seized with a somewhat severe attack of pain in the right side. The pain disappeared, and he was fairly comfortable for about a week; after that it returned, and assumed a paroxysmal character. He was admitted to the hospital about three weeks after the onset of his illness, with the physical signs of effusion at the base of the right lung. The question was what was the nature of the fluid. The mode of onset of the illness did not suggest that he had had acute pneumonia, which as I have mentioned is the usual antecedent of empyema in an adult. But I thought it would be better to put in an exploring needle so as to determine the nature of the fluid. That was accordingly done, and we drew off a few ounces of purulent fluid. One naturally, but erroneously, as it turned out, concluded that it was an empyema, and I asked my surgical colleague to operate in the usual way. When the surgeon opened the pleural cavity he found it empty, but on exploring further by passing a trocar and canula through the diaphragm he there struck pus, and it appeared that this proceeded from an abscess on the upper surface of the liver, which was no doubt secondary to the attack of dysentery which had occurred a year before. On looking back at the history of the case, the points of diagnostic importance were that the pain was more persistent, and sweating was more marked than is usual in a case of pleurisy. These features, together with the history of dysentery, might have suggested the nature of the disease. That case may help you to keep in mind the possibility of mistaking liver abscess for pleurisy.

I must now say a few words with regard to prognosis in cases of pleurisy with effusion, a matter I have already incidentally referred to. The immediate prognosis is certainly extremely good. You will remember I called your attention to the small number of deaths which have occurred at a general hospital like St. Thomas's, and this in spite of the fact that in hospital practice one gets a large proportion of serious cases. In general practice you will see very few cases in which uncomplicated pleurisy with effusion comes to a fatal termination. Death occurs usually from some complication, such as pericarditis, or from tuberculosis affecting other parts of the body, especially tuberculous meningitis or general tuberculosis. The termination in tuberculous meningitis is perhaps the most common, and I have seen a number of cases in the course of years in which this complication has occurred. The first case which I saw came somewhat as a surprise to me, for I did not then realise the close association of tubercle and pleurisy. The patient had been a fellow student with me, and one who was regarded as particularly strong and wiry. He was attacked by what seemed to be ordinary acute idiopathic pleurisy. After a time he was tapped, and clear serum was withdrawn; but he did not get well, and

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a certain degree of pyrexia continued. It was suspected that the fluid might have become purulent, and he was tapped again, but only a little serum was withdrawn. After a few weeks, one morning he told me he saw double; and one found, on examining him, that he had paralysis of one external rectus. This seemed a somewhat puzzling complication, and it did not then occur to me, as it would now, that he might have tuberculous meningitis. I know I thought of syphilis, and I remember asking him about that as a possibility. Next he complained of headache, then other nervous symptoms developed, and finally he died. We found that the pleurisy was tuberculous, and that he had a somewhat chronic form of tuberculous meningitis.

But although immediate death in pleurisy is on the whole exceptional, the frequency with which phthisis develops later on is a very important point to remember in prognosis. The view one takes with regard to the effect of pleurisy is of practical importance in relation to life assurance,* and I consider that a person who has had an attack of pleurisy with effusion is to some extent a damaged life. If you have a candidate for assurance who is weakly looking and has a bad family history, and there is also a history of his having had an attack of pleurisy, I say unhesitatingly that, though you can find no evidence of disease in the lungs, such a person should be rejected as an undesirable risk for life assurance. If you have a person who is apparently quite robust, but who has had an attack of pleurisy with effusion, he may be accepted, but he should unquestionably be rated up. I think, for instance, that a person of thirty years of age with such a history who is applying for life assurance should have an addition of about seven years.

Next we come to the treatment of pleurisy with effusion, and I want especially to impress upon you the great importance of fresh air and good food. If you look at your text books you will find little or nothing is said about general hygienic treatment. Pleurisy is not a disease in which "nature means to kill," and what we dread are not the immediate but the more remote effects. It only shows how slowly medical practice alters, that even at the beginning of the twentieth century such treatment as vigorous blistering, leeching, cupping, and the administration of antimony and calomel should be recommended in some quarters as suitable for the early stage of the disease. This so-called vigorous treatment is only calculated to prolong the duration of the illness and to predispose to future complications. If you look upon cold as the cause of pleurisy with effusion, no doubt you will shut your patient up in a warm room, close all the windows and the door, and make every effort to prevent the slightest breath of air from coming near him. But if you look upon it as a tuberculous affection you will open the windows widely, and let the air of heaven and the sunshine in freely. You must see for yourselves that the air of the patient's room is as pure and sweet as the air outside. It is not sufficient to open the windows, for the room may still be stuffy if the register of the grate be closed and the door kept shut. See, too, that the patient is not made too hot by too much wrapping, but has sufficient covering to prevent him feeling cold, and let him have wholesome nourishing food and plenty of it. This is the plan of treatment which I strongly urge for your adoption in such cases. I am convinced that open-air methods applied to pleurisy with effusion shorten the illness, alleviate the symptoms, hasten the recovery, and diminish the risk of later tuberculous complications. The first case which I treated in this way was that of a young woman at St. Thomas's Hospital who had not been getting on well in the general ward. The patient's bed was wheeled out on to the balcony of the ward overlooking the river, and she was kept there night and day. The result was extremely good, and the patient at once felt the good effects. After four days the fever, which had lasted nearly three weeks, subsided, the appetite which had been in abeyance returned, and she made a rapid and excellent recovery. It is probable that if the ordinary plan of treatment in the general ward of the hospital had been continued she would have remained ill longer and have had a more tedious convalescence.

For the relief of pain in the early stage there is nothing better than linseed and mustard poultices. If the pain be very severe one may apply a few leeches locally, and administer morphine hypodermically or internally. A little later in the case sometimes it is a great comfort to the patient if

* Hector Mackenzie, "On Pleurisy with Effusion in Relation to Life Assurance," 'Proc. Life Assur. Med. Off. Assoc.,' 1897.

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you strap the side with belladonna strapping. The belladonna strapping combines the relief which is afforded by the fixation with the anodyne effects of the belladonna. The cough, when painful and unattended with expectoration, should be checked or relieved. A simple linctus, liquorice lozenges to suck, or warm milk to sip, may do all that is required. A remedy of recent introduction which I have found useful in this kind of cough is heroin, a morphine derivative, which may be given occasionally, in the form of tablet or linctus in doses of one-sixteenth to one-twelfth grain.

In cases of moderate effusion the absorption of the fluid may be helped by the application of iodine tincture or solution over the back and front of the affected side of the chest. Some recommend that you should give diuretics and hydragogue purgatives in the cases of pleurisy with effusion, in the hope of assisting the absorption of the fluid; but such remedies are, in my opinion, as a rule, distinctly harmful, although useful enough where there is a passive effusion, the result of a failing heart. The treatment of pleurisy with effusion should be sustaining, not depleting. The ordinary drugs which one finds useful in pulmonary tuberculosis may be given in cases of pleurisy, such as creosote and guaiacol, or their carbonates or valerianates. A recent guaiacol derivative, thiocol, has the advantage over the others of being freely soluble and practically tasteless. Some who are impressed with the rheumatic origin of pleurisy give salicylate of soda, but there ought to be clear evidence of rheumatism before you adopt that line of treatment.

The next point is the question of aspiration. It is scarcely necessary to say that I do not believe in aspirating every case of pleurisy with effusion. I think aspiration should be done, first when you are in doubt as to the nature of the fluid, and second where the effusion is large or causing serious discomfort to the patient. If the amount of fluid be large or the patient's breathing be embarrassed, I should say always aspirate. But if the amount of the fluid be moderate only aspirate where you are in doubt as to its nature or where the patient is not getting on satisfactorily. I need not remind you how important it is to have your trocar and canula perfectly aseptic, and to render the patient's skin free from micro-organisms at the site of exploration. Bear in mind, however, that aspiration or even exploration with the needle is not altogether free from danger, and although such a catastrophe is very rare, death occurring during or soon after this seemingly safe and simple operation has been recorded in a number of cases. I remember some years ago one of my colleagues had a sad experience of this kind, a patient of his dying very shortly after exploratory puncture made in the out-patient room. It was suspected that there was an encysted collection of fluid at the left base, and two punctures were made; but the first was dry, and only a little blood was removed by the second. Suddenly the patient passed a large quantity of urine, turned pale, and fell backwards. Respiration ceased, and the heart beat rapidly and irregularly. Artificial respiration was maintained for half an hour. The unfortunate patient survived for three days, during which time he remained unconscious and had several convulsions. A post-mortem examination was made, but there was nothing found to explain the fatal termination. A similar case occurred under one of my colleagues at St. Thomas's, the patient being a child of seven. In another case, also at St. Thomas's, a child of eight suddenly died from syncope during exploratory puncture of lung. My friend, Dr. A. E. Russell,* in a recent paper on the subject, thinks that the sudden syncopal symptoms are due to afferent impulses conveyed along the vagus nerve to the medulla, and attributes the unconsciousness and convulsions to cerebral anæmiá, brought about by the extreme lowering of the blood pressure. Therefore never make an exploratory puncture in your surgery or consulting room. When you explore or aspirate let the patient be in the recumbent position, and always take care to have stimulants at hand, and be ready with your hypodermic syringe to inject strychnine should there be any signs of cardiac failure. Should syncope occur resort at once to artificial respiration. Another complication which has sometimes been observed to follow aspiration is pulmonary œdema, shown by the expectoration of large quantities of albuminous fluid which occurs usually about an hour after the operation.

If you find fluid to be present, the next question is, how much should you withdraw? If the fluid be of the ordinary sero-fibrinous character I would

* 'St. Thomas's Hospital Reports,' vol. xxviii, 1899.

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not recommend your withdrawing the whole quantity that it is possible to remove. If you withdraw a portion Nature apparently is as a rule able to deal with the remainder, which is gradually absorbed. If you withdraw the whole the fluid sometimes rapidly reaccumulates; and we must also bear in mind that the effusion to some extent serves a useful purpose by separating the inflamed surfaces of the pleura and so relieving pain. Then there is the question of repeating paracentesis. If the fluid reaccumulate to its former amount and the patient be not getting on satisfactorily, you should not hesitate to aspirate again, but do not be in too great a hurry to do so. Sometimes paracentesis has to be repeated two or three times before the patient makes a good recovery, but recovery nearly always does take place. If you find that after a time a certain residual amount of fluid is there, then graduated exercises sometimes are helpful in removing it, such exercises being employed as expand the lungs and bring into use the various muscles of respiration.

With regard to the after treatment, that is the most important of all. The onus rests with you not to prove that the disease has been tuberculous, but to convince yourself that it is not. Unless you can do this you must look upon it as having been a tuberculous affection, and insist that your patient after recovery from the pleurisy should get a thorough and complete change. Send him away into the country so that he may lead a healthy open-air life for a period of three months. In certain cases, if he has made a complete recovery, a sea voyage may be very beneficial. Examine the patient from time to time, and thoroughly satisfy yourself that his health is re-established.

There is just one matter I wish to refer to before concluding, and that is the question of testing the patient with tuberculin. In all cases where you are doubtful of the tuberculous nature of the illness I advise that the test should be applied for prognostic and diagnostic purposes during the stage of convalescence. I am of opinion that it will strengthen your hand and be of great benefit to the patient to make the test after recovery from pleurisy. If you find that the patient reacts to tuberculin, you can be much more emphatic in ordering the rest and change which I think are so essential. While if after the test has been thoroughly and systematically applied no reaction is obtained, you can give a more hopeful prognosis than you would otherwise be entitled to do.

WITH SIR WILLIAM BENNETT
IN THE WARDS OF
ST. GEORGE'S HOSPITAL.

A Case of Appendicitis.

Here is a young man who presents an ordinary case of appendicitis. He has had two attacks of the trouble, the last of which was three weeks ago, and he has not yet properly got over it, although he is steadily progressing towards the quiescent stage. What he calls his first attack occurred six months ago, when he says he was laid up for three weeks. But, as you often find in many of these subjects of appendicitis, he has, since he was quite young, been accustomed from time to time to get pain in his belly, often called colic or belly-ache, which is treated as a rule as being of no particular consequence. There is a peculiarity about some of these abdominal pains occurring in young people in the way in which they tend to concentrate themselves about the right side of the lower part of the belly. This is a point of some importance to bear in mind, because it is in such people that appendicitis is liable to occur. As I have said, this man is getting towards the quiescent stage after his second attack, and he has come into the hospital for operation. In examining a case of appendicitis at the end of three weeks from the onset of the last attack—that is to say, at a stage similar to that of the present case—nothing abnormal may be apparent, or there may be noticed a little want of proper mobility about the lower part of the belly. It is seen in this case that the right side of the lower abdomen is a little more rigid than the left. And if pretty firm pressure is made along the line between the umbilicus and the anterior superior spine, sliding your fingers from side to side, you will feel an elongated swelling. Now such an elongated swelling you will often hear spoken of as the appendix. This is a very common statement. On putting the hand on the belly, and making gentle but deep pressure in the direction mentioned, there is distinctly felt an elongated "sausage-shaped" tumour; this, however, is not the appendix. As a matter of fact, one of the rarest things which you feel in the belly in these cases is the appendix itself. This elongated tumour is not the appendix at all, and it very rarely is. Obviously

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if the appendix is so superficial that it can be felt by the finger in this manner, it ought not to be difficult to get at it. Let me assure you that the position of this tumour is no precise guide to the position of the appendix itself. The appendix here, indeed, may be curled up towards the kidney, or it may be down in the pelvis, or in other places,—it is impossible to say which. What we can feel here is not the appendix, but the muscular bands of the cæcum, which are very liable to be contracted. I suppose it is a spasm of the muscle which occurs. The contracted cæcum can be easily felt as a "sausage-shaped" tumour through the normal abdominal wall of a healthy person who is not very fat or too muscular. Of course the appendix may be in the centre of a large mass of inflammatory material, which can frequently be felt in the earlier stages of cases like this. You know that the point upon which so much stress is laid in connection with the diagnosis of appendicitis is the tenderness over MacBurney's point. There is no doubt that in the majority of cases of appendicitis there is tenderness over that spot. But there is also tenderness at that spot in many other conditions. It is good to bear this in mind, otherwise you are apt to be led astray by depending too much upon a symptom to which an exaggerated importance is attached by some people. Tenderness at that spot is found in ovaritis, in disease of the gall-bladder, in perinephritic abscess, and in several other conditions. That symptom, therefore, is not always diagnostic of appendicitis. If it is present it is a symptom which will help you a little, because you know that it occurs in appendicitis, but not in every case of appendicitis. Its absence, on the other hand, in no way negatives the existence of appendicitis.

This young man has, as I have mentioned, come in for operation, and the question now arises whether he is in a fit condition to have his operation at once or whether it should be postponed. It is still a matter of some doubt in the minds of surgeons as to the period at which it is desirable to perform the operation in what is called relapsing appendicitis. Is it ever justifiable for a patient to be operated upon after the first attack? If not, how many attacks can be with safety permitted before operation is deemed justifiable? Opinions differ a good deal upon this point. Personally, I am bound to say that I strongly feel that if the first attack is a typical one, the sooner that the appendix is removed the better, the quiescent state having of course been awaited in the absence of urgent indications to the contrary. In no case should any patient be advised to allow himself to go on after a second attack without having the appendix dealt with; this, however, is not the universal opinion of surgeons generally, even now. Some will tell you that there is no justification for the operation until the patient has had three or four attacks. But I think it is a good rule not to let the patient have a second attack if you can help it.

With regard to the operation, it is almost impossible to tell what the difficulties in removing the appendix will be before you set about it. No matter how simple the case appears to be from the outside, no matter how near that tumour which you feel and which you fancy is the appendix, seems to be to the surface, it is impossible to gauge in any way the difficulty of the operation. As you know, an operation for removal of the appendix may be one of the easiest operations in surgery, but it may be one of the most difficult; and it is an operation which very accomplished surgeons have, from time to time, had to give up, simply because they have either not been able to find the appendix at all, or having found it, have not been able to isolate it from the adhesions by which it has been surrounded. And so the patient has been left in the condition existing before the operation, save that some few adhesions have been severed. Remember, then, that very great difficulties may arise in some of these cases. In all operations for this condition there is one point of very great importance, viz. that the appendix should be removed through the smallest possible opening in the abdominal parietes. If the appendix is removable at all, an incision of two inches, or at the most two and a half inches, is quite enough to get it out with certainty, and generally with ease. The smaller the incision in these cases the better, because the smaller the interference with the abdominal parietes the less is the tendency to bulging and the formation of a ventral hernia. The formation of a ventral hernia is sometimes inseparable from the operation, but it should not be so under ordinary circumstances. It is a great defect in a man that he should have a ventral hernia in consequence of the bulging of a

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scar, and the main means of avoiding its formation in cases like those under discussion is to make as small an opening as possible. In 50 per cent. of the cases an opening which will just admit two fingers will be sufficient to get as much of the cæcum out as you require. With the same object in view you should never, unless you are driven by circumstances to do so, use a drainage-tube in any operation for the removal of this little piece of viscus. The use of a drainage-tube is always followed by a weak spot in the scar, and that weak spot means the initial factor in the formation of a ventral hernia. All other things being equal, you will find that a ventral hernia after the operation for appendicitis is more likely to occur in a case in which a tube has been used than in one where it has not, because the union at the point where the tube has been is, of course, not immediate, and this means weakness at that spot.

There is a point in regard to which there is difference of opinion in these operations, and that is the method of suturing the abdominal wall. It was the fact that ventral herniæ were more or less common at one time after these operations that led people to devise different plans for suturing the abdominal wall. Amongst other plans there was devised one which is popular with some people, which is called the three-storey suture. In that the peritoneum is sutured first, then the muscles over it are sutured, and then the skin and subcutaneous tissues are united over the muscles. It was thought that by that means a better scar was obtained, that it was firmer, and that altogether the repair of the abdominal wall when this plan was employed was better than when sutures were used which passed through the whole thickness of the parieties. I do not know what percentage of surgeons use the one plan, or the other, now. I have tried the triple suturing very carefully, but I have returned to the single through-and-through suture. I found that, first, the triple suture did not make a better scar; indeed, sometimes the scar under these circumstances was not so good as that obtainable by the other method, which I now use; secondly, it necessitated the burying of a large number of sutures under the skin. With regard to buried sutures, no matter how careful you are in attempting the prevention of sepsis, they are very often a nuisance, and sometimes they are a danger, because, in spite of your care, some of these buried sutures may come to the surface from time to time; and you have no conception what an intolerable nuisance it is, both to the patient and to the surgeon, if, sometimes at remote periods, sutures commence to work their way out. This remark applies to all operations. The fewer sutures you bury in any operation the better. You are never sure of them, because even if the sutures are sterile you must remember that the tissues are not always absolutely resistant; in other words, they will not always resist tension and irritation successfully. Hence they may give way, or perhaps ulcerate, becoming invaded by micro-organisms in consequence of their resisting power being too slight. These buried sutures, then, may give rise to trouble in consequence of the microbic changes affecting the tissues around which the sutures pass. It does not at all follow, because at the end of three or four weeks or later after an operation a buried stitch gives trouble, that it was septic when it was put in. It may have been perfectly sterile when it was put in, but the tissues included in it may have been invaded by an evil microbe from the patient. So the change which comes about, and which leads to the casting off of the suture, is not necessarily the result of the use of a suture which was not made sterile, but may be due to the defective resistance in the tissues, through which the suture has been passed.

There is one question in connection with these cases about which patients are generally anxious for information. The question is this: What period must elapse, after operation in an ordinary case treated in the quiescent stage, before the patient may get about and begin to resume his ordinary occupation? It is always a difficult question to answer exactly, and you must be careful about your answer; in private work, especially, you must take care that your estimate is sufficiently wide. Nothing disappoints a patient so much as to be told that he will get about sooner than he eventually does; this disappointment will be avoided if you allow a good margin in your estimate. Suppose, for an example, this patient were told that he could return to work again in six weeks or two months after his operation, he would be disappointed, as it is certain that he would not be able to do so. If, on the other hand, a limit of four months is given, and he

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possibly becomes fit a fortnight earlier, his satisfaction would naturally be considerable. Speaking generally, a man who has had an operation for appendicitis, no matter how well he feels, or how well he seems, or how tight the scar seems, should not take any exercise for at least three months. That may seem to him a long time, but I have now had a large experience of these cases, and I am sure, if you wish to do the very best for your patient, you should not let him do anything beyond gentle exercise for at least six months. At the end of that time you may let him begin to resume his ordinary occupations; that is to say, a labourer may, at the end of six months, resume his work. Under no circumstances, unless the patient is driven to it by great emergency, should there be any attempt whatever at resuming heavy manual labour for six months after the operation. Indeed, it is quite certain that a man cannot consider himself *absolutely safe for heavy work* after an operation for appendicitis for twelve months. So you may consider the important periods after operation as three months, six months, and twelve months. In three months the active man may begin to resume his ordinary occupation in a very mild way. At the end of six months he may resume ordinary work, which is not very heavy, but he must be prepared to slack off if he finds any pain or irritation about the part. At the end of a year he may consider himself sound for any purpose. The reasons for this extreme care are two. First of all, with regard to the scar; an ordinary scar of this kind takes several months to become hard, and unless it is hard it will not bear the strain of any great tension of the abdominal walls. During the time of the hardening of the scar, therefore, it is better, although it may be very inconvenient, to prevent over-straining of the cicatrix, to make the patient wear a belt of some sort. It is true that patients do not like the belt as a rule, and very often they will throw it aside in a short time; it is, however, undoubtedly better that it should be worn for three months at least. Another reason for caution about resuming ordinary habits too soon is that in many of these cases the stump of the appendix becomes adherent; further there is the condition of the cæcum, which in consequence of the interference with its normal function resulting from the appendicitis, etc., becomes very atonic, and apparently is not capable of very hard work. The gentle return to work which you allow at the end of two or three months exercises sufficient traction on the part to gently stretch the adhesions, and sometimes to separate them altogether, so that the structures concerned become loose. If, on the other hand, the patient is allowed to resume hard work of any kind, whether of amusement or occupation, within six months of the time of operation, severe sudden strain may cause any adhesions which have occurred to tear. This breaking through of adhesions may be curative, but, on the other hand, some disaster to the bowel may happen. I know of two cases in which violent exercises, one at the end of three months, and the other at the end of four months, ended in very serious disaster indeed. I am, therefore, not speaking upon theoretical grounds, but upon the evidence of cases which have been under my own observation.

One other question remains, does recurrence of symptoms identical with those caused by disease of the appendix occur after its removal? The answer to this question is in the affirmative, an interesting fact with which I propose to deal in a clinical lecture shortly. Although recurrence of symptoms after operation must be familiar to all surgeons of any experience, I know of no published cases.

We have received from Messrs. Allen and Hanburys, Limited, a bottle of a new preparation, "Byno-Cascada"—a combination of bynin with Rhamnus purshiana and Rhamnus frangula. The bitter and disagreeable taste of cascara has militated considerably against its use, but in this preparation it has been masked to a considerable extent, and its combination with bynin is an advantage, especially in the constipation of old age, or during debility following severe illness. Bynin itself has a slightly laxative tendency, and the active diastase present improves the power of digestion and assimilation, whilst the cascara and Rhamnus frangula regulate and give a healthy tonic action to the bowels. It has been found that the addition of a certain proportion of Rhamnus frangula is of use in reinforcing the aperient action of cascara after patients have become habituated to that drug, and this in combination with bynin should give the new preparation a large field of usefulness and popularity.

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A DEMONSTRATION OF CASES AT THE GREAT NORTHERN CENTRAL HOSPITAL.

By C. E. BEEVOR, M.D., F.R.C.P.,
Physician to the Hospital, and to the National Hospital for Paralysed and Epileptic.

THE first patient is a case sent to me by Dr. Tresilian, of Enfield, and it is an example of myopathy or primary disease of muscles. It belongs to the same family as pseudo-hypertrophic paralysis, and this case is the variety affecting the face, first described by Landouzy and Déjerine.

The patient is a youth æt. 17. His eyes are never quite shut, and one of the things he noticed first was that the soap got into his eyes when washing. He can elevate the upper lip, but he has more difficulty than formerly in drawing back the corners of the mouth. You will notice that he cannot purse up the lips, and when he whistles it is done with the edge of his lips. There is weakness of the orbicularis oris and also of the orbicularis palpebrarum, and some difficulty with the zygomatics. His tongue is all right. He has got practically no sterno-mastoid muscles, but you can see the omohyoid. He has some difficulty in getting up off the chair. He has extreme lordosis and arching of the back backwards. This arching of the back may be due to several reasons. It may be due to weakness of the spinal muscles, or to weakness of the glutei, or, as I think it is in this case, to weakness of the recti abdominis, which bend the spine forwards. He also has very marked winged scapulæ. That may be due to weakness of the serrati, or to weakness of the trapezii. Here, as soon as he begins to advance the arms or abduct them from the trunk, the scapulæ project; he is able with great difficulty to get the arms to the horizontal line, but not above this point. This is due to the serrati not drawing the scapulæ forwards. There is great prominence of the cervical vertebræ. He has lost the muscles which put the head forward. I think he has a hypertrophied latissimus, and his pectorals are nearly gone. With regard to his respiratory muscles, he has fair chest expansion. I will now show that the lordosis is due to absence of the recti abdominis by making him lie down on his back and then ask him to try and get up into the sitting position while I fix the thighs on the ground. You see he cannot do it properly. All the work done in the attempt is performed by the psoas and iliacus muscles, which arch the spine convex forwards, but there is no power to approximate the sternum to the pubes by means of the recti abdominis. That the lordosis is not due to the erector spinæ being weak, or to the glutei, is shown by the fact that he can bend forward and touch the ground and get up again without assistance. He gets up off the ground like a pseudo-hypertrophic case, having first to put his hand on to his knee to assist himself. With regard to the legs, there is considerable wasting of the muscles, but not so much as in the arms. He has very fair power in the extensors. His knee-jerks are present. He cannot extend or flex up the ankle, and he has some contracture in the calf muscles. There is no anæsthesia, and no trouble with the sphincters. Now, with regard to the diagnosis and prognosis of these cases. First of all, the differential diagnosis between this disease and a case of ordinary muscular atrophy is a matter of much importance, especially in the matter of prognosis. In a case of progressive muscular atrophy the first muscles to be affected are those in the ball of the thumb, the face being not affected, except in bulbar cases. The grouping of the affected muscles is very different in the present case, when the shoulder muscles are first affected. On electrical testing there is no reaction of degeneration; there is not the change which is brought out in cases of muscular atrophy due to disease of the anterior horns. There is no fibrillary tremor in this case, as there is in the spinal cord cases. The prognosis as regards life is more favourable than in the progressive muscular atrophy cases due to spinal cord disease. There is, however, no hard-and-fast dividing line between these cases and those of pseudo-hypertrophic paralysis. This type of case was first described by Landouzy and Déjerine, but it is a similar disease to pseudo-hypertrophic paralysis, only it comes on at a later age and it does not run such a rapid course. Sometimes these cases go on for many years. The great fear with this case is that he may at some time get bronchitis, and then his respiratory muscles may not be equal to the occasion. It may be that the wasting of his muscles has come to their limit. In him no muscles have hypertrophied, except perhaps one or two about the scapula, such

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as the supra-spinatus. His trapezius is almost entirely gone. What the origin of the disease I have just shown you is, it is difficult to explain. It is looked upon by some as a congenital condition; that is to say, that the muscles are not properly formed and that they remained wasted. Why the muscles in some cases are well-formed up to puberty and then waste, it is difficult to explain. Pseudo-hypertrophic paralysis usually begins at about five or six years of age, but the cases of the disease which is here shown do not begin as a rule till puberty.

The next case is that of a man under the care of Dr. Jackson, who has kindly allowed me to show him. His age is twenty-two, and there is no family history worth mentioning. It is a case of syringomyelia. Three years ago he had some stiffness about the left thumb, and gradually the whole hand wasted. Seven months ago the right hand wasted, and he had some aching pains in the left ulnar region. A week before admission he had a symptom which is very characteristic of syringomyelia, that is to say, he rested his arm on a hot oven door and burnt himself without knowing it, until he afterwards found there was a blister at the spot. He has wasting of the muscles, and this condition begins with wasting of the muscles of the hand, just as in progressive muscular atrophy. He has increased deep reflexes. His thumb muscles are flattened, and so are those of the little finger, and there is a tendency to have the "clawed hand," while there is a prominence of the heads of the metacarpal bones due to paralysis of the interossei. He has lost the power of abducting the thumb. He can just separate his fingers from one another, but only with difficulty. He has lost the abductor indicis. You will notice his hands are very blue, and they mark easily. His nails are ill-developed from trophic changes, and the hands perspire very readily. When he tries to shut up his fingers in grasping he over-extends his wrist at the same time. One cannot perform the movement of closing the fist without putting the extensors of the wrist into action; in this case the extensors of the wrist do not know that the flexors have gone, and they therefore do their part of the business as usual, and acting alone they over-extend the wrist. It is another illustration of that dictum of Dr. Jackson's, that the brain knows nothing about muscles, but only about movements. His upper arm muscles are strong and very well developed. He retains tactile sensibility everywhere, but there is some loss to painful impressions, especially over the left arm. There is, as I show you, a distinct loss to heat and cold sensation over certain areas. But in a case of progessive muscular atrophy the patient tells you right each time whether the test tube is hot or cold. I had hoped to be able to show you another patient to-night, but she is in the convalescent home connected with the hospital. She had loss to heat and pain in both arms, and in the neck and head, with the exception of the face, upwards from the second rib. This patient has increased knee-jerks, but has no clonus. The pathology of syringomyelia is that there is a cavity in the cord, in most cases in the brachial enlargement. It may be, and in most cases it is, a congenital condition. It may be due to a dilated central canal, or there may be extra cavities formed during development, especially in the grey matter and in the posterior columns. In some cases there is a glioma growing round the central canal of the cord and affecting chiefly the grey matter and pressing on the white columns, so that the symptoms are those of disease of the anterior horns, that is to say, muscular atrophy just like that in a case of progressive muscular atrophy. Associated with that you get these changes of loss to temperature but not to touch, increased knee-jerk, ankle clonus, and sometimes a spastic condition of the legs. Another point is that the patient has not got trophic changes. Joint diseases are also seen; one man I saw with Mr. Lunn at Marylebone Infirmary had dislocations of both wrists and both shoulders, and that was a case of syringomyelia, which was verified post mortem. The prognosis of syringomyelia is generally better than that of a case of progressive muscular atrophy, as regards life.

The next case is a man æt. 57, the subject of locomotor ataxy. He has some very marked sensory changes. There is no specific history obtainable. Eight years ago he had tingling and numbness of both hands, with some inco-ordination of hands, and some difficulty in walking. There was loss to touch on the ulnar borders in both arms and the soles of the feet, also loss to pain impressions in both lower limbs. During the last four years he has had some loss to pain and temperature on the whole body except the face; also lightning

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pains in the legs and some difficulty with micturition. At the present time he has unequal pupils, the right being larger than the left. There is no reaction to light, but there is to accommodation. His knee-jerks are absent. His walk is slightly ataxic, and there is slight staggering with the eyes closed and the feet together. There is loss of sensation to painful impressions. In locomotor ataxy you may have loss to touch or to pain, or to both. You will notice that when he walks he keeps a very broad base, and he has a difficulty in turning round. He experiences difficulty in touching the tip of his nose with his finger when his eyes are closed. You will notice that he calls a pin-prick a touch on the left; but on the right he can feel the pin-prick. Where there is the loss to pain there is also loss to sensation of heat and cold. He has sensory changes in the legs, but I specially want to show you the sensory changes in the body. In tabes we recognise that we are liable to have sensory changes in the legs, but you may, if you look for them, find sensory changes in the trunk also. He has some trouble with his water, and gets lightning pains in the legs, and also in the arms. In cases of tabes the upper part of the cord as well as the lower may be involved. The reason he is not so ataxic as many cases is that he still has tactile sensibility and the sense of position in the legs. His muscles are not wasted anywhere. You may say, why is not that a case of syringomyelia, as he has more loss to pain and heat and cold than the last patient. But you do not get lightning pains and loss of the pupil light reflex in syringomyelia, and there is nearly always muscular wasting. This present patient illustrates the fact that locomotor ataxy is not always such a rapid disease as one usually considers it is. He has had trouble of the kind for ten years, but he is still able to walk about I have had other cases of the same duration in which the patients have been quite able to walk, and in whom the disease has not progressed.

The next patient is a young man æt. 28, a patient of Dr. Ferrier, who has allowed me to show him. It is a case of disseminated sclerosis. There is no family history of importance obtainable; no history of specific taint. The onset of his trouble has been gradual, consisting of difficulty in walking, with his toes catching on the ground, seventeen months ago. At night time the legs started. At the same time he had hesitancy in micturition. Ten months ago he had become much worse, and the legs became stiff and aching. At the present time the chief points about him are a typical walk, nystagmus, and increased knee-jerks, with ankle clonus, extensor response of the big toe on tickling the sole of the foot, and some difficulty in micturition. The typical speech, however, is absent. You see he starts off to walk with a severe tremor in the limbs and shaking of the body. It is different from an ataxic walk and different from a cerebellar walk. He has not got very much inco-ordination in his hands. The nystagmus is of slight degree, but enough to enable a diagnosis to be made. Sometimes disseminated sclerosis is said to be confused with paralysis agitans, but there is really no likeness between the two to justify this. Disseminated sclerosis comes on before thirty years of age, whereas paralysis agitans comes on after forty-five as a rule. Further, a case of paralysis agitans is always moving, the movement being diminished, if anything, on voluntary effort; but the opposite is the case in disseminated sclerosis, for the patient is quite still when at rest, and it is voluntary action which starts the jerky movement. There is no wasting of the muscles, and no loss of power. His knee-jerks are very active. You will see the extensor movement of the big toe on tickling the sole of the foot, a condition which is not obtained in health after a person has begun to walk. This is always associated with sclerosis of the lateral columns or with the cutting off of the cerebral influence. You may obtain this in cases of Friedreich's disease, where you get sclerosis of the lateral columns and also of the posterior columns. So that while, in these cases there is no knee-jerk, the extension of the big toe shows that the lateral columns are involved. He has no affection of sensation, but he has to micturate as soon as he feels the desire to do so.

The last patient I have to show you is one I have not had an opportunity of examining yet, as she only applied for treatment to-day. She is a patient of middle age, who has been in bed seven weeks. Her legs are very painful, and there have been mental changes. There is a history of excessive indulgence in alcohol. There is some stiffness of the leg. She can invert her foot, but cannot evert it very well. There is some contraction of the tendo Achillis, due to the calf muscles

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being unopposed. In all these cases of peripheral neuritis of which this is an example you find the muscles are tender. Her right leg is not quite so bad as the left, but there is a double foot-drop. There is not much anæsthesia in the legs. In peripheral neuritis you get all four limbs affected, especially the extensor muscles of the hand and foot. She has lost the power of extension of the fingers. Why the extensors suffer more than the flexors it is difficult to say. It is probably because the extensors of the fingers are the weakest muscles in the body, and those which do scarcely any work. For instance, the extensors of the fingers simply put the fingers into position to enable the flexors to do the work, as in grasping an object.

The prognosis in peripheral neuritis due to alcohol is good if the supply of spirits can be completely cut off, and though recovery may be delayed for weeks or months, the cases usually completely recover. Hence the great importance of making a correct diagnosis and finding out the cause.

A BUNCH OF ENLARGED GLANDS SIMULATING THE OUTLINE PRODUCED BY AN ANEURYSM WHEN EXAMINED FROM ONE DIRECTION BY THE X RAYS.

F. H. WILLIAMS, M.D.

In some instances new growths under the upper part of the sternum simulate a thoracic aneurysm. It is therefore important to examine the dark area seen on the screen during both inspiration and expiration, and carefully observe its outline. If there is no pulsation of the outline seen, the shadow may or may not be due to an aneurysm. If there is pulsation, and the outline increases in size, the dark area is probably caused by an aneurysm. If there is pulsation which produces displacement but does not enlarge the outline, the dark area is more apt to be a new growth than an aneurysm.

The following case is chosen to show how a bunch of enlarged glands may give outlines on the fluorescent screen that simulate those produced by an aneurysm; also to show the importance of examining the chest from different directions in order to locate exactly the object producing the shadow cast on the screen; and, thirdly, to show the importance of considering other indications of disease besides the appearances seen on the screen.

Mr. C—, æt. 40, was referred to me by Dr. John W. Farlow for an X-ray examination. He had complained of pressure which was sometimes felt in the upper portion of the thorax. He had paralysis of the vocal cord, dyspnœa, etc. Some of these symptoms suggested an aneurysm, but other signs characteristic of the disease were wanting.

X-ray Examination with Screen.—I examined the patient with the fluorescent screen, and found that there was a dark area in the neighbourhood of the aorta, but there was no pulsation of these abnormal outlines; that there was obstruction to expiration; that the lungs were dilated to their fullest extent; that the diaphragm was low down in the chest, nearly as far as the costal border, and moved very little in expiration.

Judging by the appearances on the screen, it was difficult to decide whether we had to do with an aneurysm or a new growth, but I finally inclined to the former view, though I did not make this diagnosis; the case was puzzling, and the outlines seen on the screen, though in some respects similar, yet differed from any other case of aneurysm of the aorta that I had examined with the X rays. I expected, however, to see the patient again, get history, and take an X-ray photograph; but was disappointed in this expectation. Where the appearances are unusual, at least two radioscopic examinations should be made.

The patient died on May 10th, 1899, and no aneurysm was found.

The above is taken from a very valuable work by Dr. Francis H. Williams entitled "The Röntgen Rays in Medicine and Surgery." The volume is published by the Macmillan Company, and is profusely illustrated, no less than 391 blocks being used for this purpose. The particular object Dr. Williams has set before him is to explain the help obtainable from the Röntgen rays in medicine and surgery as an aid in diagnosis and as a therapeutic agent. The author states on the title page that the book is designed for the use of practitioners and students, and he is certainly well advised in employing the word "use," for no reader could fail to find ample material in the pages of this work of the kind and character most likely to lead to the author's teaching becoming exceedingly useful to the medical man in his every-day work. Though the author says his book is rather a report of progress than a final presentation of a growing subject, the net result of his labour is to furnish the medical man with a scientific exposition of a difficult subject in such clear and accurate language that it is as pleasant to read as it is easy to understand.

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THE CLINICAL JOURNAL,

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* *Specially reported for The Clinical Journal. Revised by the Author.*

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A CLINICAL LECTURE

ON

DIABETES.

Delivered at Guy's Hospital, June 4th, 1902.

By W. HALE WHITE, M.D., F.R.C.P.,

Physician to the Hospital.

GENTLEMEN,—We have had for some time past a very severe case of diabetes in Philip Ward, and we now have one in John Ward, and Mr. Milsom has made some laborious investigations on the urine in the one in Philip, and Mr. Brown is doing the same with regard to the case in John; therefore I thought we should be well employed to-day if we went over some points about diabetes in a clinical lecture.

The fundamental fact about a case of diabetes is that the patient excretes sugar in his urine, and for the case to be one of diabetes the sugar must be dextrose, or, at any rate, very largely dextrose, for Dr. Pavy has shown that there are often traces of other sugars present also, and no doubt that is true; but the fact remains that dextrose is by far the most important of the sugars present. Before we go further into our subject you will remember that you must be careful in testing urine to make sure that you have to deal really with dextrose there. Many people are condemned as having diabetes owing to faulty testing of the urine on the part of the doctor, when the patient really has no diabetes at all. You are aware of the necessity of being careful that your Fehling's solution is fresh; and that is the reason why you boil it before adding urine, because if it is stale a precipitate will occur without the addition of anything at all. But many other bodies beside dextrose may exist in the urine, which will cause a precipitate on the addition of Fehling's solution. For instance, uric acid and creatinin and creatin will all reduce it. But you will always find that the reduction in these cases is not the bright yellowish-red reduction of genuine sugar, but it is the dirty green

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reduction with which you are all so familiar as occurring in the wards. That does not indicate the presence of sugar. Then, sometimes bodies which reduce the copper solution much better are present in the urine, and yet the case is not one of diabetes, and usually the body which does it is glycuronic acid. Glycuronic acid may be present in the urine after the administration of many drugs. For instance, it may be present after the administration of nitrite of amyl; it may be present after taking chloral; after taking morphia; and sometimes it may be present after taking salicylic acid; and it may also be present after taking chloroform or prussic acid. I have not time now to give you instances, though I easily can, in which every one of the fallacies about the detection of sugar that I have mentioned has occurred in my own experience. One instance, to show you how sad it may be, I may tell you. I once saw a woman who was being treated with diabetic diet under the impression that she had diabetes with phthisis as a complication. She was really dying of her phthisis, and had been for some time. I could find no sugar in her urine, so I asked the doctor to let me test it with his Fehling's solution. The moment I boiled his solution an abundant yellowish-red precipitate fell, and further testing proved the urine contained no sugar. So this woman for the last two or three months of her life, although dying of phthisis, had been treated with that extremely uninteresting diet to which we condemn diabetics because her urine had been tested with stale Fehling's solution. Another thing you have to remember is that occasionally glycuronic acid will appear in the urine without any drugs being taken at all, and that is another very interesting little side point which we cannot stop to go into to-day. I once saw a patient who had some of the symptoms of diabetes, but she had no sugar in her urine, though she had an abundant quantity of glycuronic acid. You have to remember, also, that sometimes genuine sugar gets into the urine, which is not dextrose. For instance, a suckling woman will pass lactose in her urine, but she has not got diabetes.

However, having mentioned those fallacies, let us narrow ourselves down to the cases in which there is actually dextrose in the urine. But even then we have to subdivide them, and it is doubtful whether all the subdivisions should be regarded as diabetes. One or two of the things strike any of you who are fresh from physiology. The first is the appearance of dextrose in the urine as the result of damage to the central nervous system. You know that it can be produced experimentally in animals by damage to the floor of the fourth ventricle, and if you tested every specimen of urine passed you would be surprised how frequently it is present in people who are admitted to hospital suffering from concussion. Dr. Campbell, who is now at Bradford, examined the urine of such patients systematically for some time here, and at some time or another during the first few days of their stay in the hospital after the concussion many of them passed traces of dextrose in the urine. Then, too, we have people in the hospitals with tumours of the brain who pass dextrose in the urine; and, in fact, we have clinically many repetitions of Bernard's experiments of the introduction of glycosuria by damage to the central nervous system. Whether these should be called genuine diabetes is merely a question of nomenclature. Dr. Fagge was of opinion that they were better not called so, and I think so too. I think it is better to call them cases of nervous glycosuria, because probably the glycosuria is of far less importance to the patient than that of genuine diabetes. In the first place, clinically it is unimportant because, as a rule, it passes away in a few days, just as experimental glycosuria by damage to the central nervous system passes away. At any rate, it is so rare for a case of glycosuria due to nerve lesion to be of clinical importance from the point of view of glycosuria, that I think we need not stop to say anything more about it. The probable pathological explanation of it is that there is only the hurrying on of sugar into the circulation which ought to have been stored up.

The next group about which there is some doubt as to whether they should be called diabetes are the so-called gouty or alimentary glycosuria cases. This form occurs in old people, and it is very important for you to bear in mind as clinical men that nobody wears out in a particular part of the body to the same extent as another. Some of us begin to wear out early as regards our hair, and some of us do and some do not wear out early with regard to our teeth. Others do not wear out with regard to their heart until perhaps eighty or ninety years of age. Elderly people sometimes

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seem to wear out as regards their power of storing up of sugar, and late in life, at the age of about fifty or sixty, they pass straight into their blood ingested sugar instead of storing it up, as you and I do. This alimentary glycosuria is solely a failure of the body to utilise ingested sugar. It passes into the blood and so into the urine. And this sugar is purely dextrose, and in these people the copper reducing test, the yeast fermentation test, and the polarimeter all show analogous results in the estimation of the sugar, showing you are dealing with a single body and not, as I shall show you presently when talking of diabetes, with many abnormal bodies in the urine. These people do not give what we will come to presently, a ferric-chloride reaction; they have not excessive acidity of their blood, and they can, generally speaking, and, indeed, almost always, be cured by so adjusting their diet that they neither pass sugar in their urine nor lose weight. Often these patients are overdieted, and too much carbo-hydrate is taken out of the food, so that they lose weight in consequence. We only need to see that they do not pass sugar in the urine, and that they do not lose weight. Probably the fundamental differentiation of these two groups, namely the nervous glycosuria and the alimentary glycosuria, from genuine diabetes consists in the fact that in neither of the first two is there any hyperacidity of the blood, but in genuine diabetes there always is, at some period or another of the illness.

Now I want you to consider for one moment the nomenclature. I think men get confused about this. The disease which we are now about to describe, diabetes with hyperacidity of the blood, must be separated off, certainly clinically and perhaps certainly pathologically, from the nervous and alimentary forms. Dr. Pavy prefers to call the form that we are going to describe "composite diabetes," for a reason I will tell you directly. Dr. Fagge thinks that the alimentary and the nervous forms should be called glycosuria, reserving the name diabetes for the other form. I should mention here that occasionally the alimentary glycosuria cases become genuine diabetes later on.

Now with regard to true diabetes. But before beginning that, please to remember you must separate sharply diabetes from the experimental glycosuria which can be produced in animals—and in the man too for that matter—by the administration of phloretin and phloridzin. When either of these is administered there is no excess of sugar in the blood, for their action is on the kidney. In every case of genuine diabetes there is an excess of dextrose in the blood. Where does this excess of dextrose in the blood come from? It is obvious it may come from over-production in the body of dextrose, or it may come from failure on the part of the body to utilise dextrose ingested. Which of these is true it is very difficult to settle authoritatively and finally; but most people who have thought much about the subject agree that in the main the pathology of diabetes is to be sought in the fact that for some reason the body fails to utilise dextrose as it should. But although it is true that some of the sugar—and I believe by far the greater part—in the urine in diabetes is due to the fact that the body fails to properly utilise sugar, that cannot be quite the whole of the explanation, because, as you can prove in the wards, if you have a severe case of diabetes you may cut out absolutely for several days all carbohydrates from the food and yet the patient will continue to pass dextrose in his urine. And then, to show you how very complicated the problem is, I am always pointing out to you the remarkable fact which Dr. Pavy first pointed out, that you may sometimes add very little sugar to the diet and that will lead to an enormous quantity of sugar in the urine. One of his classical cases was that of a man who was on strict diet and took a pint of cider. For a long time after he took this pint of cider the sugar in his urine was enormously increased. There was a girl in the hospital under me whose quantity of sugar passed shot up and kept up a long time, and then we discovered that the only error in diet was that she had once taken a little marmalade with her breakfast. Therefore we see that while the prevailing, and probably the correct, opinion as to the pathology is that the body fails properly to utilise sugar, and hence it exists in the urine, nevertheless that cannot be the whole explanation; there must in some cases at any rate be an increased production of sugar. And Dr. Pavy believes, and you will see there is a very good reason for the belief, that the extra sugar comes from the proteid tissues of the body. They abnormally break down in such a way as to lead to the production of sugar,

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and it is because these cases of genuine diabetes have two sources for their sugar, namely the failure to utilise that which is ingested, and also some produced which ought not to be produced, that Dr. Pavy prefers to call these instances of genuine diabetes "composite diabetes" thus indicating two sources of sugar. I think that is an excellent name. Before leaving that, please notice again the pathological importance of the fact that giving a little sugar may lead to a hundredfold amount of sugar appearing for days afterwards in the urine. That makes one stop to think, and shows, as Dr. Fagge pointed out years ago, that in a bad case of diabetes sugar is a downright poison.

Now to come to another part of the subject, the fundamental point of which is that the patient is passing out of his system by means of the urine a quantity of sugar, and as a result of that he is losing a lot of energy.

If a man passes, as a bad diabetic case will pass, half a pound of sugar a day, he is losing what energy he might otherwise have derived from the breaking down of that sugar. To show that that is so I have copied out from von Noorden's classic book on the subject the figures from one of his patients. You will see how important this loss of energy by means of the urine is. Mrs. B— weighed 55 kilos., therefore her needs were $55 \times 35 = 1925$ calories. She took daily in the form of albumen 148 grammes, her albumen was worth to her 606·8 calories; she took 102 grammes of fat, and her fat was worth 948·6 calories; and she took 180 grammes of carbo-hydrates, which were worth 738 calories, making a total in all her food of 2293·4 calories. You may say she is having enough, because she only requires 1925, and she is taking 2293. But she excreted 141 grammes of sugar every day, the caloric value of which is 578 calories. Thus she was losing this quantity of energy per day. Her food was worth 2293, and she was losing in her urine 578, so she was only really getting from her food 1715 calories a day, whereas she required 1925. Thus she was short every day by 210 calories of energy. That shows you why it is so important pathologically to the patient, this loss of sugar in the urine. It is a dead loss of energy. But this failure on the part of the patients to utilise their dextrose, leading to this great loss of energy is a very subtle problem, and we have not yet got to the bottom of it. And I want you to think, in connection with it, of the following facts. In the first place, they can utilise levulose. Then there is the fact I mentioned of a little sugar leading to a large amount of sugar passed by the urine. That is difficult to understand. Another thing is, although we know that the body, for some reason or another, cannot utilise the dextrose and oxidise and break it down, yet the failure of the body generally to oxidise is not gone; it can use the fats; lactates in a diabetic are oxidised to carbonates, and benzol is oxidised to phenol. Another thing is that people who are asphyxiated and therefore cannot oxygenise well, do not, as a rule, get diabetes. Therefore, this failure to break down the sugar is evidently a very difficult failure to properly understand, and it has been suggested that what is really the matter, pathologically, with these people is not that they fail to break down the sugar through all its steps down to its lowest breaking down, but that they fail only in one particular step; but what that step may be we do not know. You will say is all this of any clinical value? Yes it is, because now that I have been able to put before you some of the pathological facts, they will enable you to explain many of the symptoms.

In the first place, we can explain the polyuria, the increased amount of urine passed; more water is passed to get rid of the sugar. Even as it is, with the polyuria the specific gravity is up to 1030 or 1040. This high specific gravity is explained by the presence of the sugar. And in passing let me remind you that we are often confronted with a patient in whom the sugar-containing urine has a low specific gravity, often due to the fact that such patients are subject to chronic Bright's disease as well as diabetes. The specific gravity of their urine, because of the Bright's disease, would, perhaps, be 1008 or 1005, but the sugar in the urine brings up that specific gravity to 1016 perhaps. So a low specific gravity with sugar in the urine should always excite apprehension, and is usually indicative of a bad prognosis. Remember that the presence of glycuronic acid does not give a high specific gravity in the urine. The wasting is clearly explicable from what I have told you. If the patient is losing every day, say, 210 calories, the energy which is short owing to excretion of sugar in the urine, the body tends to make up from other tissues, and hence fat disappears and the

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muscles waste because of this effort to make up the requisite quantity of calories from other tissues. You have also to remember that some of the sugar which passes out in the patients' urine ought to go to build up their fat and their proteids; so you see that they do not get that source for building up fat and proteids; and this accounts for some, but probably a small part, of the wasting. The breaking down a quantity of fat and proteid in their body to compensate for the loss of energy in the urine leads to the explanation of another symptom, namely, that these patients pass an excess of urea because of the excessive breaking down of the proteid which is to compensate for the loss of energy in the sugar passed in the urine. Another symptom follows from what I have told you, namely, their weakness. Another symptom is pruritus, which is often troublesome, especially about the vulva. There is sugar in the urine, and some hangs about the vulva and prepuce in uncleanly people; it remains there and ferments, and the irritation sets up the pruritus. Then, inasmuch as these people are so much below par and wasted, they are an easy prey to all sorts of infections. Diabetics bear any septic process extremely badly; they are very liable to carbuncles and boils, and are especially liable to the invasion of tubercle bacilli. That concludes the sugar part of the question.

But I have not explained, after all, the most important symptom, namely the coma. In all marked cases of coma you will find the acidity of the blood to be increased. It was increased both in the case which Mr. Milsom looked after and in the case which Mr. Brown has looked after, and I shall show you before we have finished the reasons for supposing that this increased acidity is the cause of the coma. The increased acidity is due, most people believe, to β oxybutyric acid, which is in excess in the blood in cases of diabetes; other substances which contribute to the increased acidity are diacetic acid and amido-acetic acid. Acetone is also present. All this is of very great importance, because we believe that the acidity is the cause of the coma. These abnormal substances are passed in the urine, but most of them are difficult to detect there, and hence the importance of the ferric-chloride reaction. It is believed that the diacetic acid in the urine causes the urine to assume that dark red wine colour with ferric chloride which you all know from testing in the wards. You must add enough ferric chloride to re-dissolve any precipitated phosphates. There is no other ready test for determining whether these abnormal acids I have told you of are present, and hence it is a simple and a ready test of the very greatest practical value if, as we believe to be true, the presence of these acids means that the patient is liable to have coma. If there is no ferric-chloride reaction present it is not a bad case; if there is marked ferric-chloride reaction it is usually a bad case, because the patient has got an excess of acids, and we believe that these acids are the cause of the coma, and hence such a patient is always liable to coma. A bad case may pass 20 to 30 grammes of oxybutyric acid in a day. Think what that means. The normal alkalies of the blood are the sodium phosphate and the sodium carbonate. Suppose there is a sudden flooding of the blood with acid, there is not enough sodium phosphate and sodium carbonate to unite with this excess of acid, so a fresh draw is made upon the body, and that draw always takes place first upon the ammonia, and a quantity of ammonia goes into the blood to unite with this excess of acid, and in a very bad case calcium and magnesium are called forth from the tissues into the blood to neutralise the excess of acid there. And this excess of ammonia, of calcium and magnesium, is passed in the urine, and hence it follows that if we estimate the amount of ammonia and calcium and magnesium in the urine, we have a rough estimation of the excess of the acidity. The amount of ammonia per day passed normally is rather over a gramme, but in a case of diabetes you will often find it is considerably in excess. Mr. Milsom and Mr. Brown have analysed the urine in both the cases of diabetes which we have now in the hospital, and in both of them the ammonia was considerably in excess—in Mr. Brown's case it is 6 grammes, and in Mr. Milsom's 2·6. That, therefore, shows that the acidity of the blood was considerably increased and we infer from that there is a great excess of these acids present in the blood.

Another way of getting at this question of whether there is an excess of acids in the urine and therefore in the blood is this; you distil the urine with strong sulphuric acid, and you can recover the crotonic acid, this coming from the oxybutyric acid. Mr. Milsom and Mr. Brown have

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both done this, and the urine in each case shows a large amount of crotonic acid. This is confirmatory evidence that these patients have a large excess of β-oxybutyric acid in their urine. Another way, which is employed by Dr. Pavy, is the following:—Dextrose is a right-handed body when estimated with the polarimeter, and β-oxybutyric acid is a left-handed body. If you first ferment off the dextrose from the urine and get rid of the sugar and see how much left-handed it is, the degree of left-handed rotation is approximately equal to the quantity of β-oxybutyric acid present. I have copied here from an analysis of urine in a case given by Herter the amount of bases present. You will see how in diabetes a large amount of bases are present to unite with the excess of acid in the urine. The bases in the urine in a healthy person were as follow, in grammes: K_2O, 0·9232; Na_2O, 3·881; CaO, 0·2154; MgO, 0·0806; NH_4, 0·4707 (giving a total of 5·5709 grammes). In the diabetic they were as follow: K_2O, 2·554; Na_2O, 2·445; CaO, 0·803; MgO, 0·1973; NH_4, 3·1130 (notice the very large figure which ammonium reaches in the diabetic) (giving the very much larger total of 9·1128 grammes). You will say what is the proof that this acidity has anything to do with the coma? Well we went a long way towards proof in the cases forming the subject of this lecture. Theoretically it should be a good thing to give diabetics alkalies, and when people begin to get symptoms of coma the administration of alkalies is good, but they must be given in large amounts to be efficacious for the patient. In Philip Ward the patient was given 300 grains of bicarbonate of sodium a day, and by that means we were able to neutralise the excess of acid, and I think those of you who watched the case had little doubt that the bicarbonate of sodium saved his life. The woman in Miriam too was very much improved by it, so much indeed that we hoped she was going to recover. Not only did we diminish the coma, but we saved the call upon the tissues of these patients for ammonium, calcium, and magnesium with which to neutralise the acids in the blood, and this diminution of coma on giving alkalies is evidence that the coma is due to acidity. Another reason for believing that acids are the cause of coma is that Sternberg and Grube claim to have shown that by injecting diacetic acid into animals symptoms very closely akin to those of diabetic coma can be produced. If what I have been telling you is true, another fact is explained, namely, that coma is so often preceded by gastro-intestinal disturbance, especially abdominal pain, vomiting, and constipation. Probably the reason is that one channel, namely the bowels, is no longer active, owing to the constipation, and so some acid which might have been got rid of by the bowel is retained, and hence the patient becomes comatose. At any rate this clinical fact remains, that it is very bad treatment to allow a diabetic patient to become constipated.

How the acid causes coma I do not know, but the following suggestion has been made. The sodium in the blood naturally helps to carry the carbonic acid from the tissues to the lungs. If the sodium salts are taken up with this excessive acid in the blood the carbonic acid cannot be carried from the tissues to the lungs; and it has been suggested that this causes the coma. That does not in any way affect the quantity of oxygen in the blood, and that is why a person with diabetic coma is never livid; it is simply an inability to carry the carbonic acid from the tissues to the lung. Few gas analyses have been made in such cases, but in the few which have been made the investigations have confirmed the fact that there is a deficiency in carbonic acid in the venous blood. The source of these excessive acids is almost certainly in the proteid tissues, and it is the abnormal breakdown of proteid tissues that leads to their presence in the blood and hence in the urine.

Now we have considered two metabolic processes. And there is a third which rarely occurs, and about which I can tell you little; but I may only mention it to-day because it was so beautifully shown in the patient in Philip Ward. That is, that there is sometimes in the blood a curious condition which is sometimes due to fat. Many of us saw the blood of the patient in Philip Ward whom we venesected on two occasions, a clot separated from the blood which I cannot compare in colour to anything better than to strawberries and cream. That has been observed in cases before, but it is not a common phenomenon. Of the mode of production of this curious condition of blood we are very ignorant. It is usually said to be due to fat, but we could not obtain any evidence of fat with osmic acid, although under the microscope we saw a number of granules which were quite uninfluenced

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by osmic acid. I sent the blood up to Cambridge, where Mr. Cole analysed it for me, and I have his analysis here. He reports that the blood does not, in the ordinary acceptation of the term, contain fat, because he says, "I find that the white substance is insoluble in water and salines, and more or less soluble in alkalies. It seems to be a proteid precipitated out by the presence of a fatty substance; but what the proteid was I was not able to discover. The fatty substance is not a true fat, but is apparently an ester of cholesterin with one of the higher fatty acids, or a mixture of them." So that is very interesting as showing that sometimes at any rate this fatty-looking stuff, looking like strawberries and cream, in the blood, is not due to fat in the ordinary sense of the word, and that it does not stain with osmic acid like ordinary fat does. I have looked in many books, and I find the statement copied from book to book, that fat may be found in the blood of diabetics, but I have come across a case recorded in the 'Edinburgh Medical Journal' of July, 1879, by Saunders and Hamilton, which bears much upon ours. In it there was just the same condition as we saw in the blood of our patient. But the authors say that in that case not only were fat globules present which would stain with osmic acid, but also there was a granular precipitate to be seen under the microscope which would not stain. And that suggests that in our case we had the granular precipitate which Mr. Cole analysed with the result I have read out to you, but we did not have the fat globules. There is no doubt, I think, that in the case recorded by Saunders and Hamilton fat was there, because the patient died and sections were cut, and the capillaries and vessels were found to be full of fat globules which stained with osmic acid, and there are chromo-lithographic plates in the paper showing this. But I am able to tell you nothing as to how it comes about that this condition appears in diabetes. So extreme was it in our case that we saw a strawberry-and-cream fluid circulating in the retinal arteries and veins, producing a condition the like of which I have never seen before. I had a water-coloured drawing, which I show you, made of the retina, but it is not quite a faithful representation, because the artist has depicted the blood as rather too yellow.

You will, perhaps, ask me, are there any post-mortem appearances characteristic of diabetes? In a certain number of cases the pancreas is found diseased. Here are figures from Guy's which will interest you on the point. During fourteen years here we had 6708 post-mortems, and among those post-mortems the pancreas was diseased in 142, that is to say, 2 per cent. In nineteen of those 142 cases the diseased pancreas was associated with diabetes. In sixteen of those nineteen the pancreas was small and atrophic, in one there were pancreatic calculi which had led to atrophy of the gland, and in one there was a large pancreatic cyst, and in the last the pancreas was very large and indurated. So there is quite a considerable number of cases of pancreatic disease associated with diabetes, and as every body else who has worked at the subject has found out, far the most common pancreatic condition to get with diabetes is a small, hard, fibrous, atrophic pancreas. It is easy for you to remember that this condition is to the pancreas what contracted granular kidney is to the kidney. I took the number of fatal cases of diabetes during the same fourteen years that I took these cases of pancreatic disease, and I found we had four times as many fatal cases of diabetes as we had cases of diabetes which showed pancreatic disease. So we got a definite disease of the pancreas visible to the naked eye in a quarter of our fatal cases of diabetes. But that figure is, in all probability, far too low, because in every case only the naked-eye appearance is recorded. Another point of view, as showing the importance of the pancreas, is that there were only three cases showing the small atrophic pancreas in which diabetes was not present, and in one of them the atrophy of the pancreas was due to pressure of a large mass of growth, and in another the patient was very wasted indeed, so that the wasting of the pancreas might have been part of the general wasting. In the third, a woman who died soon after admission, no examination of the urine was made, so she might have had diabetes for all we know. These figures are confirmed by those in Berlin. Hausemann, analysing the figures from the Berlin Pathological Institute for ten years, found diabetes without pancreatic disease eight times, with no mention six times, with disease of pancreas forty times, again showing the importance of the pancreas. And of these forty cases thirty-six were examples of simple atrophy, thus confirming our results,

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three were called fibrous induration, and one "a complicated case." The pancreatic diseases which Oser, writing in Nothnägels 'Encyclopædia,' mentions as associated with diabetes—you will remember we saw atrophic pancreas, pancreatic calculi, and a cyst associated—are fatty degeneration, indurative pancreatitis, pancreatic calculi, carcinoma, cyst, hæmorrhage, necrosis, and fat necrosis. So I think you will agree that clinically there is no doubt that disease of the pancreas is frequently associated with diabetes. It is not due to any want of secretion of the pancreas being poured into the intestine, because it does not follow in animals in which the pancreatic duct is ligatured, nor is the diabetes due to any disease of the nerves in the neighbourhood, for it does not follow in animals if you transplant pancreas from its normal situation to under the skin. It has been suggested there is an internal secretion poured from the pancreas which is constantly preventing glycosuria in the normal individual, and that is a view taken by Tuckett in a paper in the 'Journal of Physiology,' in which he suggests there is constantly being absorbed by the intestinal lacteals a body which is tending to make us all diabetic, but that the internal secretion of the pancreas is constantly antagonising this. The other obvious view is that the internal secretion of the pancreas in some way aids the complete breaking down of the sugar, so that when this internal secretion is deficient we get diabetes. Nobody knows which view is true. Before leaving this part of the subject I ought to remind you that it has been suggested that the essential parts of the pancreas that have to do with the internal secretion, the alteration of which causes diabetes, are the islands of Langerhans.

I have told you the principal facts about the pathology of the disease, and shown you how the symptoms are associated with it. And now for two or three minutes I will speak about the treatment. With regard to the treatment, I have told you about the alimentary form of glycosuria. There you have to reduce carbo-hydrates in the patient's food so that he will not pass sugar in his urine. You must not cut down the carbo-hydrates too much, or else the patient will lose weight. You have to find that point in the diet in which sugar will not appear in the urine, and yet the patient will not lose weight. You may say why stop the sugar if all that is the matter is that he takes in too much and passes too much out? Clinical experience is quite clear as to the advantage of such a course, and the reason of the advantage is probably that as it is an abnormal thing for the body to have to get rid of sugar in the urine, it upsets the balance of the patient's economy. And these patients are sometimes, but not always, obviously out of sorts when they pass sugar in their urine, and they are better when you stop it; they are not seriously ill like the composite diabetic cases, because the sugar is often quite accidentally discovered, and it may be so slight as not to necessitate any particular diet for it.

Now we will pass on to the treatment of the severe form. You might think that as the patients lose a lot of sugar it would not be bad to give them a lot of it. But you must bear in mind that, as just mentioned, it is probably a great strain upon the body to pass a lot of sugar in the urine, because it is an altogether abnormal process to get rid of a quantity of sugar. We are not built to get rid of a quantity of sugar in our urine, and you would not be giving the patient anything he could utilise if you gave him more, and you would be enormously increasing the strain on his tissues to get rid of the excessive amount which you gave him, and, further, we have seen that there is strong evidence that in diabetes carbo-hydrates may be an actual poison. We have learnt by clinical experience that these patients are very much better if you diminish the amount of carbo-hydrates in the food. But you have to remember this, that all diabetics can probably utilise some sugar, especially levulose, and the mistake is often made of cutting down the sugar too much. It is necessary for the doctor to feel his way, and to find the amount, particularly of levulose, which you can allow each patient. And while the patient is at the same time gaining weight and feeling stronger, and the ferric-chloride reaction of the urine is diminishing, the patient is gaining ground whatever diet he is on. You must remember that you cannot prescribe a uniform diet directly you see the patient. You have to find out in the case of each individual what you can allow in the way of carbo-hydrates, bearing in mind you want to keep up his strength and weight, prevent coma, and diminish the ferric-chloride reaction in the urine. It is only by

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frequent examination and much discretion you can decide in each case how much, if any, carbo-hydrates you can allow.

But there are also several other practical hints which I want to direct your attention to. Every one is agreed that you can be too strict, and that many patients lose weight because the doctor does not allow them that small amount of carbo-hydrates, especially levulose, which they can utilise. I have already pointed out the importance of the fact, however, that a slight increase in the amount of sugar, may lead to a great increase in the sugar passed in the urine. You must particularly remember that, although diminishing the ingested carbo-hydrates leads to a diminution of acids in the urine, and also to a reduction in the ferric-chloride reaction, yet suddenly taking all of them out of food may sometimes produce coma. Therefore you might say, "Is not it good, therefore, if the patient is comatose, to give sugar?" It has been tried, and I have tried it myself, but no good result has followed. Nor is it any good to give pancreas to diabetics. We have tried it and it is useless. Another little thing which I want to point out to you is that there is a very common and serious error met with frequently, and that is that diabetics can take toast with impunity. I cannot trace that falsehood—for that is what it is—to its originator, and I do not know who started the notion that after toasting bread it is no longer a carbo-hydrate, but it is very widely spread among people that a diabetic can take toast. There is no justification for it, and until you can toast bread so that you can make it no longer a carbo-hydrate the diabetic must not have it. I would recommend you, before you give diabetic foods, to occasionally pour iodine on samples of them. Some manufacturers of diabetic foods, like some manufacturers of other articles, frequently adulterate the foods with starch. I have had specimen foods sent to me containing much starch. Another point in the treatment of diabetes is that fats are particularly useful. Many a diabetic is given too much meat and not enough fats. They bear fats very much better than anything. I need not go over the various foods which are given in these cases. The substitutes for bread which contain little carbo-hydrate are gluten bread, soya beans, cocoanut flour, almond flour, plasmon, and these are those frequently met with in England; abroad rice albumen, wheat albumen, and milk albumen are used. The cheapest of all these is cocoanut, and the poor can buy cocoanut in large quantity, grind it into powder, and make cakes of it at home. If you wish to give levulose that can be given as such. We tried a few years ago giving dahlia tubers, as the starch in them is inulin and is converted into levulose. Their use was only fairly satisfactory, as they were difficult to cook nicely. If you wish to give milk without its carbo-hydrate you can mix cream with albumen, water, and add saccharin to taste, as was recommended by Williamson. For some reason which we do not understand, few things are worse for a diabetic than worry. Many a time coma has been brought on by the excitement of a journey, or by some nervous worry, or something of that sort. Every diabetic should be placed in a condition of quiet and content. The question is often asked whether such patients should go to a health resort. There is no evidence that the composite diabetic benefits by such a change. The people who do improve at health resorts are those who are suffering from alimentary glycosuria. These people have their bowels kept open at such establishments, and some of the sugar is kept out of their foods, and they get on very well.

With regard to drugs, they are of secondary importance compared with diet. Opium and codeine can restrain the passage of sugar in the urine, and it is very striking, and must have struck you all, that although the diabetic person is particularly liable to coma, he bears opium well. Probably the next best drugs to opium for restraining the sugar in the urine are the salicylates.

If the coma is only a little drowsiness, dissolve 300 grains of bicarbonate of sodium in a pint or two of milk and let that be drunk during the twenty-four hours. If coma is well marked, transfuse a solution of bicarbonate of sodium into the cellular tissue of the breast or axilla, or throw it up the rectum; the patient ought to get, in a severe case, 500 grains of sodium bicarbonate in a day.

The prognosis must be quickly summed up. You cannot give a prognosis in any case until you have seen the effect of treatment. The prognosis depends on how quickly you can get the sugar which is in the urine under control, how quickly you can get the weight increased, and how quickly you can get rid of the ferric-chloride reaction. You know that frequent coma is bad, that young subjects do badly, and you know, too, that people who get phthisis, or any complication, do badly. With regard to the question What makes the pancreas diseased when it is affected? we know nothing of that. There is slight evidence that the disease is hereditary, but it is not very much so; and it is not known to be transmitted directly from the mother to the fœtus. The fœtal urine in a case where the mother is diabetic does not contain sugar, but the liquor amnii does.

We have only been able to touch on the fringe of many branches of this fascinating subject, and I trust I have told you enough to interest you in it, but as I have already exceeded my time I must now say no more.

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A CLINICAL LECTURE ON FRACTURES IN THE NEIGHBOURHOOD OF JOINTS, AND ON SPRAINS AND DISLOCATIONS.

Delivered at the City Orthopædic Hospital, June 11th, 1902.

By E. NOBLE SMITH, F.R.C.S.Edin.,
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DISLOCATIONS and fractures in the neighbourhood of joints, and even severe sprains, are frequently followed by permanent impairment of articular movement and by deformity.

Much may be done to prevent or minimise these unsatisfactory results, and I propose to discuss the methods of treatment which I have found useful.

The principles of treatment I am about to advocate have received but scant attention in this country in the past; and even in the present day, although a certain number of surgeons have adopted such principles partially, the advantages of the methods referred to are not universally recognised, and are by some surgeons entirely opposed.

It is twenty years since I wrote in advocacy of frequent passive movements of joints near fractures.* It is six years since I published the results I had obtained by using early strapping, early movements, and massage in the treatment of fracture of bones, and it is fifteen years since I published the good effect of daily movement after reduction of dislocations. I then described the principles which I thought should generally be applied in the treatment of human dislocations, as follows:—(1) Allowing as short a time as possible to elapse before the reduction of the dislocation is attempted; (2) avoidance, as a rule, of making such attempts until the patient is under an anæsthetic; and chiefly (3) daily movements of the joint, commencing soon (twenty-four hours, perhaps) after reduction.†

I shall describe some of the cases here referred to presently, but I allude now to the dates of these observations to establish the fact that I was one of the first in this country to advocate the reforms which have since been adopted by several other surgeons.

Whether we are concerned with cases of fracture, of dislocation, or a combination of both, the less the parts are disturbed in a preliminary examination the better. It is, of course, very desirable that the surgeon should satisfy himself generally as to the nature of the injury, but his examination need not involve detrimental movements of the injured parts.

While a patient is conscious he cannot, during the manual examination by the surgeon, control entirely the action of the muscles which surround or act upon the seat of accident, and every movement of a fracture or displaced bone not only causes severe pain to the patient, but induces the muscles to act automatically. This spasmodic action tends to obscure the nature of the accident, and may render a clear diagnosis of the case very difficult.

Under such circumstances it frequently happens that this preliminary examination fails to give the surgeon sufficient information, and he has ultimately to place the patient under an anæsthethic before he can make out exactly the condition with which he has to deal. Moreover, if the case be one of dislocation, the proper reduction of the displaced joint generally requires an anæsthetic for its performance. Also some fractures are much better "set" under an anæsthetic than without one.

This being so, it is much better to administer the anæsthethic before a detailed examination is made; that is to say, when the surgeon has determined by his visual observation of the parts, and by a gentle handling, that a serious injury has occurred, involving probably fracture or dislocation, he will best serve the interests of the patient and his own convenience by delaying a more detailed examination until the patient is insensible under chloroform, and the muscles can offer no resistance to manipulation of the parts.

Then, again, by waiting until the patient is anæsthetised the setting of the fracture, or the reduction of the dislocation, can be effected at once while the patient remains unconscious and the muscles are temporarily paralysed. Thus the more exact diagnosis of the injury, and the replacement of the bones in a correct position, are

* 'Surgery of Deformities,' London, Smith, Elder and Co., 1882.

† 'Brit. Med. Journ.,' January 7th, 1888.

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carried out at one and the same time, in place of a very painful and harmful examination and a subsequent attendance of the surgeon to "set" the parts.

Another very important point is involved. Any undue handling and consequent disturbance of injured parts leads to increased effusion of blood and serum, and further rupture of tissues from movements of irregular fragments of bone in the case of fractures. The consequence is that when the parts have to be "set" much swelling may have occurred, and the surgeon may have less chance of effecting a perfect re-adjustment of the injured parts.

Of course there are many cases of fracture in which the bones can be so readily placed in a good position that no surgeon would think of using an anæsthetic, and even in dislocations immediate reduction may sometimes be effected with so moderate an effort that the necessity for chloroform would not arise. Also the accompanying circumstances should have an influence. When, for instance, an anæsthetic is not procurable, or not without long waiting, we are justified in making greater efforts to reduce a dislocation or a fracture. My contention is that in nearly every case of dislocation, and in those fractures which cannot be quite easily reduced, there are distinct advantages to be gained by placing the patient under an anæsthetic before a prolonged examination is made.

The use of the X rays is invaluable in the diagnosis of injuries to bones and joints, but we must not forget that what we can see is not a real view of the parts but merely a shadow of them.

It follows that the result of an examination by this means is likely to give an erroneous idea of the condition, and we have yet much to learn of the process before we can thoroughly rely upon it to give us exact information about fractures and dislocations.

However, while we must not discard the older methods of forming opinions as to the nature of any paricular injury, we have in X-ray photography an extremely valuable means of adding to our information; a means which ought, if possible, never to be neglected. I use the word photography because I have little faith in an examination with the screen alone.

There is always a certain amount of swelling of the soft parts after reduction of fractures and dislocations, and it is this swelling which so seriously complicates what may be a comparatively simple fracture or dislocation.

The swelling is caused by an effusion of blood and serum into the interstices of the injured tissues. It constitutes a species of dropsy of the part, and, when once it has occurred, there is great difficulty in reducing it. In the 'Atlas of Histology,' which I published (in conjunction with Dr. E. Klein) in 1880, I have shown some fibres of connective tissue separated by the extravasation of such fluid, and as this specimen was magnified over 450 diameters, it will be realised how intricate is this permeation of plasma. The most serious result of this extravasation is that if it remains in a stagnant condition it becomes in time organised, unless, indeed, it becomes septic, breaks down, and forms an abscess.

This latter result is fortunately rare, but organised thickening of the parts from effusion of blood and serum was at one time quite a common occurrence after one of the injuries we are considering, and even at the present day the proportion of stiff joints following these injuries is considerably larger than it should be.

A severe sprain, in respect to liability to effusion of fluid, resembles very closely a fracture near a joint, or a dislocation.

In fact, we may consider that in the latter injuries the parts are sprained as well as fractured or dislocated.

A sprain may be considered a dislocation without permanent displacement. Therefore, in discussing the treatment of fractures near and into joints and dislocations we ought to be also considering to a certain extent the treatment of severe sprains.

In all such injuries our first consideration (after placing the injured parts in a normal position) should be to prevent effusion of fluid at the seat of injury.

The swelling which takes place is often so severe that surgeons have feared to apply firm support to the affected parts directly after an accident.

Keeping the limb raised to the level of the body or even higher is a most important and ancient procedure, and should not be neglected. Bandaging has been also carried out, but with great caution, because it has so often proved to be

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harmful by interfering with the circulation, or by causing irritation. All text-books enjoin great care in bandaging to ensure the evenness of the pressure, and many good surgeons deprecate the use of bandages entirely. Of course, when splints are applied some amount of pressure must occur, but then cotton wool or other soft substance is interposed between the splint and the limb to limit this direct pressure; moreover, I am now referring to the application of a bandage to the skin itself before the splints are put on. It is more than twenty-five years ago that I began to use adhesive plaster in the treatment of sprains instead of the ordinary roller bandage.

The procedure is as follows. The sprained joint is raised as far as possible above the level of the body to encourage gravitation of blood and effused fluid, and, if the parts will bear it, a little gentle pressure with the hand—practically slight massage—is applied, working towards the body. After about five minutes or more of massage the adhesive strapping is applied, commencing (in the case of a sprained ankle) from the base of the toes and advancing towards the calf, to about three or more inches above the ankle.

Now, the method of application of strapping is of the utmost importance. If the strapping is not put on with absolute evenness, or if it is drawn too tight, it may do a great deal more harm than good.

This procedure should not be entrusted to a nurse, however skilful she may be; it is essentially a matter for the surgeon to carry out.

The chief point in the use of strapping in this way is that it should be *laid on evenly without making the least effort to pull it tight.* That is, there should be no attempt to constrict the part; no attempt to lessen the swelling by pressure.*

If these instructions are followed out no harm will result. At least, I have never experienced any but a satisfactory effect, and I have used strapping in this way in a large number of cases during the past twenty-five years. On one occasion I had some fear as to the result. It was the case of a man who had injured his ankle while hunting. He had not only severely sprained the joint, but had also crushed it between his saddle and a gate-post, and the parts were so injured that I thought the soft tissues would mortify. That they did not do so was chiefly due, I believe, to the use of the strapping. If any further effusion had taken place I think that such an unfortunate result would have occurred. However, he made a good recovery.

After applying strapping the surgeon must watch his case closely, and if any feeling of tight pressure is realised by the patient, the surgeon must remove the strapping and re-apply it. I have invariably found that the patient has experienced great comfort immediately the application has been made, and the removal and re-application of the plaster has generally been desirable in a couple of days or sooner, or perhaps later, on account of shrinkage of the swelling.

Thus we may go on changing the strapping every few days while the swelling decreases. Under the most favourable circumstances the strapping will have been applied before any appreciable swelling has occurred, and then probably no removal may be required for a week.

At the periods of renewal some gentle massage should always be given, the degree depending upon the exact condition of the parts.

Hot fomentations are not, as a rule, desirable directly after a sprain has occurred, because they tend to cause more effusion, but a few days later, when the ruptured vessels have closed up, hot applications may act beneficially in conjunction with massage.

After a few days' rest, or even at the commencement of treatment some movement of the joint should be carried out and the joint may be moved passively while the strapping is in place. This movement should, as a rule, be passive, but in the less severe sprains the patient can sometimes walk about (assuming that we are still dealing with a sprained ankle) very soon after the strapping has been applied.

Having met with great success with this treatment in the case of sprains, I came to apply the same principles when dealing with dislocations, chiefly in respect to early movements and massage. In dislocations I would urge that early and frequent passive movement of the joint is the most important part of treatment.

On February 18th, 1887, I was called to see a lady æt. about 35, who had injured her right

* In the 'Lancet' of December 8th, 1900, a letter appears, written by me, upon this subject, giving many of the above details of treatment of sprains.

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elbow-joint from a fall, and it was apparent at first sight that either fracture or dislocation had occurred.

No attempt to move the joint was made until the patient was under ether, when I found there was a dislocation of the forearm backwards. This was easily reduced, as the muscles were temporarily paralysed by the anæsthetic, and the arm was then bandaged upon a rectangular splint. Two days later I removed the splint and commenced passive movement of the joint, replacing the splint. This movement was repeated again three days afterwards, and then daily. Ten days after the accident I could move the patient's arm to almost complete flexion and complete extension. Three weeks after the accident the splint was discarded entirely, and three weeks later the patient was well. There was perfect freedom of movement and no pain or swelling. This lady was then able to return to her duties as a pianist, and had no further trouble.

I have carried out the same method in all similar cases which have come before me since that date, in many of which strapping from the first and massage have been applied in addition to repeated passive and active movements.

To show the result of the early-movement treatment after dislocations, I will record an instance which occured several years ago of dislocation of the shoulder in a mare. I gave the animal chloroform without removing her from the place where she had fallen. I reduced the dislocation by bringing the leg forward, grasping the hoof and throwing my weight suddenly backwards in a line with the horse's body. The head of the bone, which had produced a projection plainly visible upon the outer part of the scapula, receded into its socket with a loud snap, audible to my friends and others who stood around. Upon the animal's recovery to consciousness she was able to get up and to walk, with an occasional trip, about a mile to some stables, whereas before the reduction she could not put the foot of the lame leg to the ground. She was taken to town by train the same evening, and was walked from the railway station to her stables, about two miles distance. She was led up and down the mews every day until, on the seventeenth day after the accident, she was driven in a light brougham round Regent's Park. Two months after the accident I rode her to Wimbledon Common and jumped her over some small fences, and the following season hunted her again. No further trouble occurred with the joint.*

We know how commonly stiffness or absolute fibrous ankylosis occurs after dislocations, results which may, I believe, be generally prevented by the treatment here advocated. The same principles of treatment will be found very effective in restoring action to a joint which has stiffened from a want of early movement after dislocation, but under such circumstances the treatment has to be much more strenuously applied, and the breaking down of the adhesions already formed has generally to be effected first.

The treatment of fractures by massage and early movement of adjacent joints has not been readily received by the majority of surgeons. This is not, perhaps, surprising. Absolute fixation of the fractured bones has for ages been urged as necessary, and any treatment which seemed to jeopardise such fixation was necessarily looked upon as questionable. The fact that massage and passive movement of the involved joints could be effected without harmful disturbance of the fracture was not readily realised, and also the fact that if any slight movement of the fractured fragments upon one another were to occur it would not necessarily be detrimental to good union. Certainly in some cases of long-delayed union of a fractured bone it was customary to make some movement, such as causing the fractured ends to rub together, with the object of stimulating bony repair, but that was looked upon as a sort of necessary evil, and after such movement the parts were as carefully fixed as before. The result, however, of absolute fixation has been almost invariably to cause a certain amount of stiffness of the limb from organisation of effused plasma among the muscles and tendons round the joints. Muscles and tendons become adherent one to another to such an extent that no subsequent force can entirely overcome the stiffness. The nerves also often become involved in the matting together of the parts.

The early use of strapping limits the effusion massage and early movement maintain the function of the muscles, and diffuse into the circulation whatever extravasated fluid remains. These

* 'Brit. Med. Journ.,' February 15th, 1886.

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remedial methods also prevent that wasting of muscles which so often results from long-continued fixation; they further and consequently prevent the pain arising from the involvement of nerves in the adhesions.

One of the most painful and complicating accompaniments of fractures is spasm of the muscles. Such spasms not only cause great immediate suffering to the patient, but are also apt to displace the fractured bones after the most accurate "setting" and careful fixation. The gentle application of surface massage will almost invariably remove this symptom.

In fractures, accurate diagnosis and accurate "setting" are of the first importance. Too much trouble cannot be taken in replacing the broken fragments as far as possible into their natural position. For this object alone an anæsthetic is of the greatest service. It relieves the patient from all pain, and not only prevents spasmodic action of the muscles, but also relaxes these muscles from their ordinary tonicity. Our next object is to keep the bony fragments in their natural position, and, to do this, splints of some kind are often necessary. Upon the other hand, there are many fractures which do better without any splints at all When we have to use splints they should confine the parts no more than is absolutely necessary, and should be easily removable for the application of massage and movements. Splints must be used when we cannot by any other method keep the re-adjusted bones in position, but here it may be remarked that re-displacement of fragments is very often brought about by muscular spasm, and this spasm may generally be removed by gentle massage. It therefore follows that many cases which, by the old plan of treatment, absolutely necessitated the use of such fixation apparatus, may now be dealt with entirely or partially without them.

I will give a few examples to illustrate this subject.

Intra-capsular fracture of the neck of the thigh bone is best treated without any splint. We do not expect bony union, so we should aim at obtaining a useful limb with fibrous union. In this injury muscular spasm is a very painful and troublesome symptom, and it is here that massage has its most valuable effect. Careful surface massage may be commenced from the very first and carried out daily, or twice a day. From the second day careful passive movements may be permitted. This treatment not only comforts the patient and helps repair, but it also prevents that wasting of muscles which so frequently follows this injury. As regards movements, they should consist of flexion and extension alone at first, and rotation should not be practised until a week or fortnight after the injury. But in all cases massage should precede the movements.

Fracture of bones of forearm above the wrist—Colles' fracture—used to be almost invariably followed by stiffness of the wrist and fingers, and it has been proved that such stiffness was due almost entirely to adhesions around the joints.

Twenty years ago I wrote as follows: "Much might be done to prevent the occurrence of these stiff joints by frequently applied passive exercise of the parts. If the fracture is situated very near to the joint, the difficulty of employing passive motion is, of course, proportionately great, and Colles' fracture of the forearm has been considered to be one in which no movement of the hand ought to be allowed.

"The writer has, however, devised a plan for making the part of the pistol-shaped splint which is in conjunction with the fingers movable opposite the respective joints, and so allowing passive movement to be made without disturbing the setting of the fracture.*

"A splint has also been invented by Dr. Carr, an American surgeon, for the treatment of Colles' fracture, and the results attained from its use are reported to be very good.

"It is said to 'relieve the patient from much discomfort and disability, allowing him to use his fingers and thumb, and thus, by the excellent position obtained, preventing the stiffness frequently occurring after this fracture.'"†

(At the time I wrote the above remarks the treatment by passive movements was generally considered outside the range of orthodox practice, and I was quite severely criticised for advocating such a treatment. Now surgical views are changing in this respect, and the fact that such measures are of enormous value in helping forward repair and

* It is now generally recognised that flexion of the wrist is a much better position than the adduction produced by the old pistol-shaped splint.

† 'Surgery of Deformities,' 1882.

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preventing stiffness and deformity is becoming recognised).

Another very useful splint allowing movement of the wrist and fingers in Colles' fracture is that devised by Dr. Alexander Gordon. This splint combines the principles of fixation of the fractured bones in a scientifically correct position, while it allows freedom of movement to the wrist and finger-joints. In cases of this fracture, movement of the wrist may be commenced five or six days after the bones have been "set," gentle massage having been applied from the first. In moving the wrist the surgeon should grasp the fractured part with one hand to steady the injured bones, and gently flex and extend the wrist with the other hand. The fingers also should be moved several times a day by the patient or nurse. This should be done while the splints are in position, the hand being free, massage always being carried out before movement.

Some surgeons have discarded splints entirely in the treatment of Colles' fracture.

In fractures about the shoulder-joint splints may often be discarded with advantage, a sling supporting the forearm and a cap to the shoulder sufficing in many cases.

Fractures about the elbow-joint (in which massage and early movement are particularly useful) may also be treated without splints, or with splints which do not involve the absolute and prolonged fixation commonly considered necessary.

These brief notes upon the management of particular fractures must suffice to illustrate one of the principles of treatment which this lecture is meant to advocate, a more detailed account being left for a subsequent lecture.

In 1895 I had become so convinced of the soundness of the treatment by strapping, massage, and movements, that I determined to apply it in dealing with fractures involving joints.*

I was encouraged in this course by the writings of M. le Dr. Lucas-Championniere, of Paris, who had just published an extensive work upon this subject.†

On November 15th, 1895, I had a fall in which I sustained a Pott's fracture of the left ankle, and I thought that I could not have a better case in which to try what was in this country to some extent an experiment, but which I had advocated in 1882.

The following is a copy of a report of my case (after exhibition at the Medical Society of London) which appeared in the 'British Medical Journal' for February 15th, 1896.

"Treatment of Pott's Fracture by Early Strapping, Early Movement, and Massage.

"Mr. Noble Smith related that on November 13th, 1895, he fell and fractured both tibia and fibula, the tibia just above the internal malleolus, and the fibula just below its centre. The foot was turned completely outwards, the fragments, including the internal malleolus, being separated from the shaft by more than an inch. The fracture was reduced immediately, and strapping applied to the foot and ankle within half an hour, before any appreciable swelling had taken place. The strapping was laid on without any attempt to produce pressure, and was re-adjusted almost daily according to the tension, which, however, was but slight. The strapping was discarded upon the seventh day. The ordinary back splint, supporting the leg, thigh, and foot, with side pieces, was applied from the first. At the end of a fortnight plaster-of-Paris splints were substituted. Very gentle movement of the ankle-joint was kept up almost from the first. Massage was begun slightly during the first week, gradually increased, care being taken to avoid disturbance of the fractured bones. On the twenty-first day he could bear the weight of his body on the foot of the injured leg for a moment. On the twenty-third day he could walk with caution. On the thirty-second day he could flex and extend the foot to within a third of an inch of the sound one; and on the thirty-fourth day he gave up crutches, walking with two sticks. On the forty-second day he walked two miles with sticks, and the following week he went hunting."

Since that time other surgeons have taken up this subject, and in the 'Lancet' of February 8th, 1898, an editorial article dealing with this plan of treatment referred to the fact of my having called attention to the method two years previously. The following is a part of this article:

"Of all the evil consequences of fractures of the bones of the limbs none is more common or more painful than the stiffness which is so often met with after the repair of the injury to the bone. This stiffness is due to one or other or to both of two causes—the existence of adhesions between muscles or tendons and the injured bone, or the development of adhesions within the affected joints owing to injury to these joints at the time of the fracture. Extra-articular adhesions are the more common, but of the occurrence of intra-articular

* Adhesive strapping has been used, I believe, by some surgeons for many years in cases of sprain, but not, so far as I am aware, in cases of fracture.

† 'Traitement des Fractures par le Massage et la Mobilisation,' Paris, 1895.

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adhesions as a sequel to fractures close to joints there can be no doubt. All surgeons have recognised the great value of skilled massage—rubbings and movements—in the treatment of this stiffness. Under its influence the fibrous bands seem to melt away and the range of movement daily grows. In severe cases it has to be supplemented by free passive movements under an anæsthetic, but even then the treatment is prolonged and painful, and tries both the patience and the endurance of the patient. On the principle that prevention is better than cure, surgeons have been slowly feeling their way to the earlier application of massage, in the hope that by moving the joints almost from the first adhesions will be prevented from forming, and the patient will enjoy a useful and painless limb as well as a strong one when the splints are laid aside. Two illustrations of this practice at once suggest themselves. In all fractures near the elbow-joint it is now a common practice to move the joint gradually more and more freely almost from the outset of the treatment. Again, in the treatment of Colles' fracture of the radius the deserved popularity of Carr's splint is chiefly due to the fact that it affords no hindrance to the daily—or even more frequent—movement of the fingers and thumb of the injured hand. This course of procedure has become more and more popular and trusted. Some surgeons, however, have gone still further and recommend that massage shall be employed from the very outset, and they maintain that thus used it is not only a powerful preventive of adhesions, but that the fractures unite more kindly under its influence, and the troublesome pain so often experienced in the early days after a fracture will subside more quickly than if treated in any other way. . . . Some two years ago Mr. Noble Smith called attention to this subject at a meeting of the Medical Society of London."

Beneficial as massage and early movements are in the treatment of fractures, a certain degree of caution is necessary. The effect of massage should be distinctly soothing and comforting to the patient, and any adverse symptoms, such as severe pain, would counter-indicate this treatment. Movement also should be exercised only so far as the patient can comfortably tolerate it. If this treatment is commenced early, the joint can be moved with ease, and after a few moments the patient will probably recognise a beneficial influence. So long as there is no septic condition of the parts, the remedy, if carried out in this gentle manner, cannot produce harm to the general system. It is said that they might carry blood-clots or septic material into the circulation, and it has even been stated that septic pneumonia has followed the use of these remedies; but, on the other hand, it should be noted that septic pneumonia has occurred too often when the fractured limb has been kept in absolute fixation. There are evil results from the pressure of bandages and splints, and ischæmic abscesses have probably been caused or influenced by these appliances, thus showing that there is no method of treatment that may not be followed by some unsatisfactory complications. Personally I think that there can be no doubt that the treatment I have advocated is as little likely to produce any deleterious effects as any kind of treatment, and that, upon the whole, it is the safest and best method we can employ.

The use of massage after injury to a joint is contra-indicated or requires modification when there is neuritis, or tuberculosis, or gout, or a septic condition of the parts.

We meet with a variety of deformities of limbs, and impairment of movement in joints, caused by non-union of, and bad union of fractured bones, as well as by the effects of the organised extravasations of blood and serum referred to in the preceding remarks. These unfortunate conditions may, of course, result from a want of accurate "setting," or from want of proper attention to keep the bones in apposition afterwards, but we should not be too ready to assume such to be the case. The most skilful surgeon may find insuperable difficulties in getting perfect results in a complicated case; the time between the accident and the attendance of the surgeon may have been detrimentally prolonged; the health of the patient may be defective; or the restlessness of the patient or other accidental circumstances may have prevented the best results.

Many modifications in treatment of these cases have been introduced in quite recent years, and one routine and uniform line of action is by no means generally recognised as yet. The question of cutting down upon a fractured bone to remove clots, and perhaps also to adjust fractured fragments more accurately, and sometimes to unite the bones by wire, is still a question for discussion; but I would here remark that if the treatment by strapping, massage, and early movement were more regularly adopted at the outset, the necessity for such operative interference, or rather any question regarding it, would far less often arise.

However unfavourable may be the result of the primary healing of an injured joint or bone, it is seldom if ever that something cannot be done to relieve unfavourable results at a later date. Irregularly united bones can now be divided and reset with safety; stiff joints may be released without much trouble to the patient; and even when irregularities occur which it is not thought wise to try to alter radically, assistance may be given to the patient by carefully devised and skilfully applied apparatus. In this last respect a large field is open to the ingenious surgeon, who, in giving his energies to this item of treatment, should try to emancipate himself from too much dependence upon the instrument maker.

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*Specially reported for the Clinical Journal. Revised by the Author.

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SOME CLINICAL ASPECTS OF ARTHRITIS.

A Clinical Lecture delivered at Westminster Hospital, May 13th, 1902.*

By W. H. ALLCHIN, M.D., F.R.C.P.,

Senior Physician to the Hospital.

GENTLEMEN,—I propose to say something to you concerning some aspects of joint affections, a large subject dealing with a region which is on the borderland between those artificial divisions of our work which we call "medicine" and "surgery," a region in which both physicians and surgeons meet. I intend to refer more particularly to those aspects of the question that come especially into the hands of the physician, those cases, in short, which the surgeon has not the opportunity of dealing with from the point of view of treatment, and that do not require any surgical interference for diagnosis. Of course the subject of arthritic affections itself, even with the limitations I am now putting upon it, is necessarily a very wide one, and it is only some cases of arthritic disease that I propose to consider within the limits of this hour, those, namely, of which there happen to be in this hospital at the present time several extremely interesting illustrations.

The first point which one naturally would consider is, what are the conditions with which we find joint affections associated? At the stage in our work at which we have here arrived, we endeavour to get as far as possible away from mere tabular statements of disease and symptoms, or from looking on disease in those artificial ways, such as almost necessarily have to be employed in teaching beginners. In fact, I think one would not do wrong in saying that very much of the early dogmatic classification which convenience compels in respect to our first studies has to be thrown aside, and we have to endeavour to forget much of it when we are

* Being the first of a post-graduate course.

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actually dealing with disease from a more advanced and responsible point of view. And it is more the habit of generalisation, and endeavouring to look at disease from the aspects of its general features rather than from those more or less artificial limitations, which almost insensibly lead students to regard illness as separable into many hundreds of distinct maladies between which there is very little connection. We know perfectly well when we get into practice that very many cases do not conform to text-book descriptions, nor even to the appearances which we meet with in the wards, and that we have to take a far wider view of disease. Therefore, in putting before you, to begin with, anything like a tabular statement of the conditions with which arthritic affections are commonly associated, I must ask you to clearly understand that it is only for the purpose of our immediate convenience, in order to give some general notion as to the states of ill-health which may be associated with a joint lesion. These may be divided into four main groups:

I. *Trauma.*

II. *Infection:*
- Septicæmia, pyæmia (puerperal).
- Scarlet fever.
- Gonorrhœa.
- Acute rheumatism (chronic rheumatism).
- Rheumatoid arthritis.
- Influenza.
- Typhoid, measles, dysentery.
- Tubercle.
- Syphilis.
- Leprosy.

III. *Diathetic:*
- Gout.
- Hæmophilia.
- Rheumatoid arthritis.

IV. *Neurotrophic:*
- Tabes dorsalis.
- Syringomyelia, etc.

In the group of cases attributable to infection I have included only the most important, namely, septicæmia and pyæmia, including the puerperal state, scarlet-fever arthritis, gonorrhœal arthritis, and the arthritis of acute rheumatism. Influenza, also, which is not an infrequent cause of joint affections, and the somewhat rarer but still occasional occurrence of joint states in association with typhoid fever, measles, and dysentery. Tubercle is one of the commonest of all causes of joint diseases. There are also syphilis and leprosy; the former not only in the hereditary form, but also as a manifestation of the later stages of the acquired malady. For the next group I have used the term "diathetic," using a word which is convenient for present purposes, though perhaps not altogether strictly defensible. In that class there is gout, the condition of joints found in hæmophilia, and I have also included rheumatoid arthritis, which I also put among the infections. It is also likely that many cases of "chronic rheumatism," so called, come within this same category. Lastly, there is that group which, in our present state of knowledge—or of ignorance, whichever you like to term it—we call "neurotrophic" conditions, that is to say conditions which are associated with definite and very obvious lesions of the central nervous system. First in that group there are the joint affections which complicate tabes dorsalis, syringomyelia, and those ankyloses of the joints, especially of the hands and feet, which follow lesions to the main nerves of the limbs, which have been described more particularly by Mr. Bowlby. There is also another very curious class of cases which one meets with sometimes, in which occur transient arthritic attacks directly associated with migraine. Now, I do not pretend that that list is either absolutely complete or absolutely correct; its purpose is only to put before us the main conditions with which we find joint states associated. You will notice I have put rheumatoid arthritis amongst the infections, and also under the heading of diathetic. The reason for that you will see presently when I am speaking more particularly about that condition of disease, of which there are several marked illustrations here.

Looking for the moment at these conditions collectively, the first thing which strikes us in reference to our present subject is that the occurrence of joint affections is by no means a necessary phenomenon in association with any one of those diseases; that every one of those maladies which is set forth there may run its course to cure or the reverse without the occurrence of any joint affection; that the joint affections are common in varying degrees of frequency amongst those diseases, but that in no one case is it absolutely an essential feature of the disease that the joint should be affected. Even in the case of acute

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rheumatism our extended views as to the nature of that affection lead us to regard a joint affection as by no means necessary, and certainly not the only manifestation of rheumatic infection, though it is one of them, just as the cardiac state is another, and there are many who believe, and they are probably correct, that the cardiac phenomena of acute rheumatism are the more constant. This is the first fact which we have to remember, and it suggests that if the joints are liable to be affected, but not necessarily so, in a large number of affections differing in other respects very considerably from one another, that the responsibility for the incidence upon the joints must be upon the arthritic structures themselves. In this connection I may remind you that trauma has been definitely shown to favour the development of tubercle in a joint, and it may be that injury or exposure, however slight, may be the determining factor in other arthritic lesions. Mr. Lucas has explained the much greater frequency with which the left knee and wrist are affected in the gonotoxin arthritis of infants by the greater exposure to which the left side is subjected as the child lies on the left arm of the nurse. But a very interesting and, as far as I know, unanswerable question which applies to this condition of disease as well as to any other is what is it that determines the locality of morbid manifestations? Why should certain regions, whether joint or bone, or skin or mucous membrane, be the particular structures in the body which exhibit the characteristic lesions? Why should certain structures be involved in one disease and other structures in other diseases which, in a sense, may be diseases which bear on the face of them some very close association and similarity? That is an unanswerable question at present, as I say; we are unable to state in any way definitely why it is that certain causes of disease produce effects in this, that, or the other particular tissue or organ. In respect to this special proclivity of joints, the various explanations of a very partial character that have been put forward are certainly, to my mind, not either satisfactory or conclusive. One of these is that the joint is the situation from which the toxins, or the virus—using that word to mean both the organism and its toxins,—may be eliminated, and that at the site of elimination the irritant produces an inflammatory result. That may be one way of looking at it. We see something of the same kind in the case of the kidney, which, in the eliminations of poisons from the body, whether organismal or not, is apt to suffer as the result of the passage of the poisons which it is concerned in eliminating. It may similarly be that the joints are the sites of elimination. It may be also, as has been suggested, that the joints, bearing the relationship which they do to the lymphatic system, and being, as the synovial sacs are, large lymphatic spaces, may offer a convenient opportunity for the elimination of those poisons. Further, it may be, as has been thought, that, as in the joints, the tissues are not very active, but are tissues containing a relatively small proportion of living elements (such, for instance, as cartilage), that in those situations the blood- and lymph-currents are slower, and thereby offer greater opportunity for the elimination of those poisons which are in the circulating fluids. Whether any one of these views be absolutely correct I cannot say; but certainly neither is quite satisfactory as an entire explanation of why the joints particularly suffer in these maladies. We have to accept it as a fact that they do.

I should now like to make a few more general remarks upon these points, because, if what I say will be of any use, it will be less, I think, in the mere description of the phenomena, which you can obtain in very much clearer language in the text-books, than in giving you a more comprehensive and general notion, so as to enable you to take a wider view of the subject. If we look over that list of maladies with which joint affections are associated, there is one thing which occurs to us with respect to the distribution of the arthritic lesions. In several of them it is usual to find a number of joints affected. It may be that a preference is shown for the larger joints, as is the case more commonly in acute rheumatism. In rheumatic fever or acute rheumatism it is usual to find that a patient, by the end of the attack, has suffered from an affection of many of the joints of the body, and certainly the larger joints, such as those of the knee, shoulder, elbow, and wrists, while probably the smaller joints, such as those of the fingers and toes, escape. On the other hand, there are conditions in which, although the attack may at first affect several joints, the number is probably more limited, and the attack more or less definitely becomes concentrated upon one joint particularly,

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or perhaps two. That I will show you illustrations of in one case particularly; and you frequently see such a condition as that in gonorrhœal arthritis. It sometimes occurs that, as a result of the gonococcal infection, one joint only suffers, but the history shows that several have suffered in many instances, although the incidence of the disease chiefly falls upon one of them in the end. Again, in others the disease may affect one joint and one joint only throughout. That, occasionally, is the condition of things which we meet with in tubercle, or, as I have said, in gonorrhœal joint affections. It is rarely, or one may practically say never, that one sees only one joint affected in acute rheumatism; but in gout and hæmophilia it may be one joint only which is affected. There is therefore a very considerable variation in the distribution of the arthritic lesions, even amongst those conditions where the cause is of a similar kind, as of an infective character — some infections preferably affect many joints, while others preferably affect but one or two. Some affect particularly the larger joints; some particularly affect the smaller ones. It is usual to find that true gout affects one joint only, perhaps through the person's entire life, or, at all events, for many years, and that a small joint and not a large one. In the case of most infections, in tabes dorsalis, of which there is a particularly fine illustration in the ward, it is the large joints only which are generally affected. As a direct result of tabes probably the small joints are scarcely if ever affected, though this particular patient does show some affection of the small joints, an interesting condition to which I shall refer again presently.

With regard to the duration of these conditions, some of these affections are essentially acute and even transient. It is usually the case in an attack of rheumatic fever that the patient suffers from the joint affection, possibly for a few hours only, rarely for more than a day or two, and then another joint is affected, the joint first involved clearing up. You see that especially in children, in whom the arthritic lesion is most transient. One joint may be excessively painful in the morning, and later on may be practically painless, whilst some other joint which previously may have been comfortable or quite painless may be the seat of very considerable pain. On the other hand, there are others of these affections which are essentially chronic, so that when once the joint has become affected, when once it has exhibited any manifestations of disease at all, the condition does not leave it, but remains and progresses in degree. Or again, there are some of them in which the condition begins in one joint, as you will see in this case of recent rheumatoid arthritis, and gradually in the course of its progress has become spread over several. Again, there are a certain number of joints which are rarely if ever affected. For instance, the sterno-clavicular, the temporo-maxillary, and the joints between the ossicles of the internal ear are seldom involved, and the same may be said of some of the spinal joints. But an exception to that statement occurs, particularly in one of these diseases, which we will call for the moment rheumatoid arthritis, of which you will see illustrations here, in which there is considerable fixity of some of these joints.

With respect to the degree to which the joint structures are affected, there again we find a very considerable difference prevailing in these several forms of arthritis. In some cases, for instance, of acute rheumatism, and even in some cases of suppurative arthritis, as may occur in the course of septicæmia and scarlet fever, you may find after the inflammatory effusion has cleared up that the joint is as good as it was before. The person suffers from an ordinary attack of acute rheumatism, and after recovery the joint shows no sign of damage; it is as good as it was before, so far as one can judge, and no depth of structural change has taken place. The synovial membrane alone has been involved. In several cases the synovial membrane may be extremely thickened, so much so that it never returns afterwards to its normal state. Such a condition one commonly sees in tuberculous joints. Again, the morbid changes may go further and involve the cartilages, and in fact the affection may begin in the cartilages, such as is commonly the case in rheumatoid arthritis, and then the joint is more severely and deeply involved. Again, the bone may be involved as a further stage in the damage which occurs in the joint; or the joint may be invaded from the bone, such as occurs in many cases of tuberculous disease, and such cases, as a rule, are more acute in character than those which commence in the synovial membrane. In syphilitic affections of the joint, especially in hereditary

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syphilis, and sometimes even in the later forms of arthritis associated with acquired syphilis, the joint is involved by extension from the bone; and in exceptional cases there are those affections of the joints due to extension of mischief from bursæ which communicate with those joints. Much of the alteration that takes place in chronic arthritic affections is due to changes in the periarticular structures, the capsule, ligaments, and adjacent tendons and fibrous bands. Most commonly they become considerably thickened and infiltrated with fluid, which largely contributes to the swelling of the region; in contrast to this, these structures may be very much reduced in amount from absorption; this is often very marked in rheumatoid arthritis, and is well illustrated in one of the cases before you. Another feature which is worth keeping in mind with respect to such a grouping of joint affections is the comparative—I will not say absolute—freedom from suppuration which occurs in many of these forms. You do not find suppuration occurring in gout, nor in tabes dorsalis when there is the joint complication of that affection. Nor do you find suppuration occurring in simple ordinary acute rheumatism, or in rhematoid arthritis, or in chronic rheumatism. But in septicæmic cases and scarlet-fever arthritis, and sometimes in gonorrhœal arthritis—leaving aside influenza, typhoid, and measles, which are rare,—tubercle, and syphilis, there you find suppuration frequently takes place. There is a good deal of difference, then, as regards cases of arthritis in this respect.

If we view some of the common symptoms which joint affections exhibit, there is also much variety in their manifestation. Take the symptom of pain to begin with. There is a singular absence of pain in some of these affections, and a marked and intense pain in the case of others. And the degree of pain is by no means proportionate to the extent of the damage that has taken place in the joint; perhaps one might even say it is the reverse. I will show you a case in which there are joint affections, in tabes dorsalis, in which disorganisation of the joints has proceeded as far as it is possible for you to see a joint go; yet the patient suffers no pain, and never has had any pain in connection with the joints. They may be handled and manipulated without causing the patient any discomfort. On the other hand, there is the acute pain which is associated with acute rheumatism, or the perhaps still more agonising pain which is associated with acute gout, when the structural changes are inconsiderable. Even in cases of arthritis due to the same cause there is a vast difference in respect of the amount of pain in different patients. In tuberculous joints, for example, there is the greatest variety. Many cases of tuberculous disease of the joints run their course without any pain whatever. A child with a tuberculous hip may be able to go about; it may limp perhaps to some extent, but that is rather from the physical disability of the joint than from any pain associated with it, and yet there may be considerable tuberculous disease present. The next child with the same disease may suffer considerable pain. So the presence or absence of pain becomes, as in the case of most diseases, as regards its diagnostic and prognostic import, very uncertain and very variable, and requires the greatest judgment to estimate properly.

Next with regard to swelling. The amount of swelling may vary from something very considerable indeed to a scarcely appreciable fulness; and the damage to the joint as regards the deformity which it produces is also extremely variable. You will see that in one or two cases here the degree of deformity which a joint has undergone in consequence of the affection is extreme; in others it is very little noticeable. No doubt the degree of deformity in a great measure depends on the extent to which the joint structures are involved, and the nature of the change which has taken place in them; and not only the amount of destruction of tissue that has taken place in that joint, but at the same time the amount of new-formed tissue that has been developed contribute to the result. In acute rheumatism an effusion of fluid takes place into the joint, and under satisfactory circumstances that clears up, and the joint is as well as it was before. In other cases suppuration takes place in the joint, and there is considerable destruction of cartilage, possibly even of synovial membrane, or the bones may be invaded, resulting in considerable destruction, so that the joint at the end of the process would be less in regard to the amount of the material in it than before the disease started. But there are many other cases in which, alongside with the destructive changes, there has

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been a very extensive production of material in which, whilst bone has been destroyed in one situation it has been formed in another part of the joint. That is particularly the case in rheumatoid arthritis, so called. It is also the case in those forms of joint found in tabes. You will see in this specimen of that condition that there is an enormous formation of bone in some places, whilst at the same time there is very considerable destruction at the ends of the bones.

The osseous change may lead in the course of time to ankylosis and fixity of the joint, or, as in rheumatoid arthritis, to a false ankylosis from interlocking of the bony outgrowths. In the knee or elbow, the seat of the characteristic alterations of tabes, the greatest mobility of the bones may exist, producing what has been termed the "flail-like" condition. The ends of the bones denuded of cartilage, and the osseous outgrowths or deposits in the capsule and ligaments will give rise on manipulation to various grating sounds, just as the thickened periarticular structures cause creaking on movement.

Associated with the arthritic changes, and very much in proportion to the extent of those changes, is found wasting of the muscles connected with the joint. Sometimes also there is evidence of neuritis in the neighbouring nerves.

I now propose to show you several cases, bearing in mind some of those more general features which I have alluded to, which I think may be of some use in giving you a comprehensive notion of these joint affections as a whole.

The first is a simple case of traumatic joint in a girl. Some years ago she met with an injury to her leg, which resulted in a violent wrenching of her knee. Recently she has sprained it again. There is a thickening of the structures around the joint, which is simply the result of the trauma. So far as physical examination of the joint goes, it is very difficult to distinguish between it and two joints I will show you in other cases, which exhibit, as far as the appearance and physical examination of the joints are concerned, substantially the same thing. It is the history which enables us to know the difference. If you look at the knees of these next two girls you will see a similar condition of things, but the history which they give is different. The joint in this patient has been very much larger than it is, and it is much less painful than it was, but the mischief is apparently limited to the knee-joint, as in the previous traumatic case. But when her illness commenced it began with an affection of several joints of an acute character. Finally, it has more or less settled down into this one. There is every reason to suppose it is a gonococcal affection of the joint, though I am not absolutely certain. There is in this, as in the former case, the same periarticular thickening, and there is evidence of considerable effusion, and there is also more heat in the joint than in the former.

The condition of this next patient is a more difficult case to give a name to. You see in her a similar swollen and somewhat painful knee, and she has other joints, especially the ankles, also swollen, but not to the same extent. The duration of this case has been much longer, and my colleague under whose care she is thinks it is more properly to be regarded as rheumatoid arthritis. My chief object in showing these three patients is to present to you how a very similar condition of lesion may be determined in the joint, although the causes are different, viz. from trauma, from inflammation consequent upon a gonococcus, or infection, and from some cause, possibly microbic, which leads to rheumatoid arthritis.

The next two cases illustrate rhematoid arthritis about which I wish to speak rather more fully. This woman is æt. 35. Her illness began at eighteen and a half years of age; the other girl's illness began at ten years, she being now æt. 21. These patients are both suffering from what at present we include under the same designation, though they manifest a little difference from one another. I ask you to notice in connection with this older patient the extreme deformity and distortion of the legs, their eversion and abduction, which is common in extreme cases of this disease. Also notice the evidence of trophic change as regards the impaired quality of the skin, its extremely glossy appearance, which you may regard as a neurotrophic lesion. I invite you to feel those joints. There is not very much swelling about them, on the contrary the periarticular structures are much atrophied, and they are more ankylosed than is commonly the case in this affection. Book descriptions often say that rheumatoid arthritis in the joints never produces real ankylosis, that it is a false ankylosis caused by the locking of bones

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together. I think that is not the case here. Her maxillary joints are also affected, as they are in the other girl. These patients cannot open their mouths as completely as they ought, and this one has a great deal of pain at the site of the articulation. If you feel the shoulder you will be able to detect the ridges of bone around the head of the humerus. I ask you to look at the fingers particularly; they exhibit in great measure the ordinary deformity which is characteristic of rheumatoid arthritis except in one particular. Notice the wasting of the small muscles. There is in both cases a certain degree of ulnar deflexion, with the hand bent to the left; but in neither case is it as much as you often see it; this condition, as you know, is explained by the unopposed action of the extensor carpi ulnaris against wasted interossei muscles; but this deformity which you see—the extension of this first phalanx and the flexion of the next phalanx and the extension of the next one—is a characteristic appearance of so-called rheumatoid arthritis. The elder patient is dexterous with these fingers, however, and she can work beautifully with them. But you will observe there is no particular swelling of any of these interphalangeal joints; the prominence of the articulations is due to more or less dislocation which has taken place, not to any formation of new matter in the joints themselves, or to thickening of the periarticular structures. There is a singular absence of that in this case; they do not conform to the common features of rheumatoid arthritis. Neither have these fingers that characteristic fusiform shape which one so commonly finds associated with this disease, in which the swelling is opposite the middle of the joint and tapers above and below. She has no cardiac lesion whatever.

Now look at this younger girl. Her condition also began in one joint, and gradually extended. It began when she was ten, and she is now twenty-two. There is considerable shortening in this leg, and that is partly because the left femur is considerably bowed. Otherwise the affection is, in the main, restricted to the extremities, and it also is manifested in some measure in her jaws; she cannot open her mouth as completely as she should. As in the previous case, there is no characteristic fusiform swelling of the finger-joints, and in neither of them is there any very excessive amount of thickening around the joints themselves. In both of them there is that very marked and characteristic swelling which you always see over those two first joints, metacarpophalangeal, which, in my experience, is one of the most constant appearances. An important question arises in connection with this disease which these cases in a measure serve to illustrate, and that is, what is the nature of this disease which we call rheumatoid arthritis? That is to say, is it microbic? There are several observations and experiments which have gone to show that this is the case, and especially those by Dr. Blaxall of this school, who has demonstrated the existence of a microbe in these joints. But I think those who have taken up that view will admit that there is some confirmation wanted before it can be put on the same platform as that on which we can put gonorrhœal arthritis or even acute rheumatism. On the other hand, there are certain other views, one of which is that this is essentially a neurotrophic lesion; that this disease which we call rheumatoid arthritis is the expression of a disease in the joints due to impairment of nerves and nerve-government over the joint. The exact nature of a neurotrophic influence is doubtful; it is probably not separated from the influence over tissues which stimulates them to their ordinary activity and normal nutrition. But some perversion of that nutritive influence appears to exist in many cases of this disease. It is not impossible to reconcile the microbic theory of this disease with the view that it may be a neurotrophic condition. We are acquainted with neurotrophic changes which take place as a result of toxins, such as in diphtheria. There is also some reason to suppose that acute anterior poliomyelitis may be an acute infective disease, and to suppose that its characteristic results might be the expression of an acute infective disease acting specially on the nervous system. This in itself would not do any violence to the views which we hold in connection with other diseases.

The relation of this so-called rheumatoid arthritis to acute rheumatism is a question of interest upon which different opinions are held. Neither of these two girls gives any history of an acute rheumatic attack to begin with; the affection began in one joint only. They also state that they had never had rheumatism before. Certainly it does not appear as if there was any attack which

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was comparable to rheumatism, and neither has any heart disease. You will find a very large number of cases—perhaps quite half of them—give no history of acute rheumatism. This disease comes on by itself, quite independently of acute rheumatism. In other cases patients have had acute rheumatism before this has occurred. But acute rheumatism is a very common malady, and this also is a very common malady in one form or another; not as severe as in one of these patients, perhaps, but in one phase or other it is far from uncommon; therefore these two may well be accidentally associated rather than having the relation of cause and effect.

The other case I want to show you is that of a woman who is now sixty years of age. When she was between thirty and forty years old she exhibited symptoms of tabes, and she illustrates now a knee affection which is known as Charcot's joint, or tabetic arthropathy. If you feel these joints you will find that the tibiæ inside these large swellings are perfectly movable; the heads of the bones are destroyed, and there is a large amount of osseous formation around the joint. That is the characteristic of the tabetic joint when affecting the knee or the elbow. Where the shoulder or the hip are affected the wasting and destruction is usually unaccompanied by anything like the same amount of coincident production. In the knee and elbow there is a considerable amount of swelling usually, as you see here. This is the finest specimen of the condition that I have ever seen. There is a certain amount of fluid in the swelling; there is also bone, and you can ascertain that there is a grating between the masses of bone in the tendons, that is to say, where it ought not to be, whereas the heads of the bones themselves are very much destroyed. That is interesting as showing in an extreme degree this condition of change. But what is rather more interesting is the condition of her hands. This condition of hand has come on within the past six or seven years. There is a very considerable wasting of the small muscles—the interossei, with thickening around the ends of the phalanges, the latter resembling very much the appearance of what is called, and perhaps properly so called in our present view of the matter, chronic rheumatism. What one means by that, or should mean, is that condition of joint change which bears a direct relation to the previous occurrence of attacks of *bonâ fide* acute rheumatism. In a vast number of cases of acute rheumatism the joints clear up; in a certain number they do not. Now and then these patients resist treatment by salicylates; they do not get well, or if they do the rheumatism very soon recurs. That acts in such a way as to leave the joint more or less damaged, more or less stiff, and more or less thickened. The changes which occur in these joints are mainly limited to the periarticular structures. The cartilages alter very little, and still less the bones. Thus they differ considerably from the changes which occur in rheumatoid arthritis or osteo-arthritis. The phalangeal joints in this patient are more like the nodular joints of chronic rheumatism, such as you see in people exposed to damp and cold and so forth, the condition to which the term chronic rheumatism may properly be applied. I here show you some skiagraphs of this condition.

Dr. J. C. Crist, in a paper entitled "Thirty-two Cases of Typhoid Fever without a Death," published in 'The New York Medical Journal,' says: If the patient is an adult, 5 grains of carbonate of guaiacol are administered every six hours, which controls diarrhœa, relieves the distended abdomen, and reduces fever. We cannot give any fixed medication for every case, but this is the treatment usually adhered to. Sometimes the dose must be diminished or omitted, but the patient is not allowed to go longer than twelve hours without guaiacol carbonate. If the patient is not able to sleep, a dose of 15 grains of sulphonal is administered at bedtime. I have rarely had to administer a heart stimulant—only in one case of my thirty-two, that of a young woman who had a relapse. The pulse reached 180 to the minute. Strychnine sulphate in $\frac{1}{20}$-grain doses was administered hypodermically every three hours, or when it was considered advisable. After the patient is convalescent and the fever is subsiding, the night dose of guaiacol carbonate is omitted or reduced, as it will lower temperature, and it may bring it below normal; a condition I have encountered, and with alarming symptoms, on more than one occasion. We do not have a great number of cases of typhoid fever in this vicinity. My thirty-two cases have extended over a period of five years, and my experience with carbonate of guaiacol in typhoid fever has been as satisfactory as the use of quinine in malarial fever.

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SOME CLINICAL ASPECTS OF RHEUMATISM IN CHILDREN.*

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ACUTE rheumatism as seen in adults makes a striking and distinct clinical picture, with the considerable fever, the violent pain flying from joint to joint associated with swelling, redness, and acute tenderness, the furred tongue and the profuse sour-smelling sweat. Such a picture is presented with extreme rarity in childhood. There the disease is of a much more varied character, much less striking in its symptoms, and more difficult in its recognition; the outward manifestations are usually mild, there being but little fever, except with the occurrence of complications such as pericarditis or pneumonia, no great joint pain, and nearly always a complete absence of the profuse sweating of adults. The mildness of the symptoms adds perhaps to the danger of the complaint, since many patients remain untreated until irreparable damage has been done. Though much less unpleasant to the patient, rheumatism when it occurs during the early years of life is a much more serious affair than in later years. We can relieve pain and abate fever, but we are quite unable to make an incompetent heart valve again capable of preventing regurgitation or to widen a stenosed orifice.

It is the liability to heart complications which forms the most important phenomenon of acute rheumatism, and we find that the earlier the age at which the rheumatic attack occurs the more likely is the heart to be affected. Adult rheumatism has, indeed, one special danger, viz. hyperpyrexia, with its usually fatal issue. Hyperpyrexia is practically unknown in the rheumatism of children, and in fact is anything but common in adults. Apart from this, the danger of adult rheumatism is the same as in the child, viz. implication of the heart, but not only does advancing age render an attack of rheumatism less probable, but also if an attack does occur the heart is less likely to be affected than in earlier years.

It is not only the valves of the heart that may be implicated, endocardium, pericardium, and mesocardium may all be affected. Of these, the endocardium has been said to be the most frequently attacked, and it is very seldom that we find a rheumatic pericarditis without an associated endocarditis, while an inflammation of the endocardium by no means implies that the pericardium is also affected. Not very infrequently the child's heart may fail quite early during an attack of rheumatism, sometimes though not usually during the first attack. These cases of "rheumatic carditis," as Dr. Sturges used to call them, frequently end fatally. They present a fairly typical clinical picture with high fever, often delirium, considerable dyspnœa and orthopnœa, and prostration; on examination a rapid and excited action of the heart is found, greatly increased area of cardiac dulness, loud pericardial friction, and an apical systolic murmur. There is very often, too, some pleurisy or lung consolidation, particularly on the left side. The increased size of the cardiac dulness is almost invariably due to the dilatation of the heart cavities, not to an effusion of fluid into the pericardium, and it is surprising how great this dilatation may be and how rapidly it may develop. After death, as one would expect, there is found extensive inflammation of both the endocardium and the pericardium, and as has been recently pointed out the mesocardium is also deeply implicated, the gravity of the symptoms being probably mainly due to the amount of damage done to the heart muscle. All cases of "rheumatic carditis" do not end fatally; recovery with a more or less permanently damaged heart may result. An interesting feature of this particular class of cases is that while the heart symptoms are so urgent the symptoms of rheumatism as ordinarily understood are inconspicuous, the arthritis disappearing or the movements of chorea ceasing. Subcutaneous nodules are, however, usually to be found. This type of case is much rarer in adults, and we but seldom in adults have the opportunity of examining the appearances of recent rheumatic inflammation of the heart, since in them death from heart disease generally does not occur until many years after the causal rheumatic attack.

* Delivered before the West Kent Branch of the British Medical Association.

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Between these urgent cases of heart failure and the cases where the implication of the heart is so indefinite that it often is only ascertained by repeated examination, there is every gradation. Dr. Lees has urged that even in adults acute rheumatism is associated almost invariably with some affection of the cardiac muscle, resulting in a weakened force of the heart and a dilatation of its chambers. Dr. Lees founds his views on the observed enlargement of the deep cardiac dulness in cases of rheumatism. Authorities, however, differ so much in their conclusions as to the "deep cardiac dulness" that one is rather sceptical of theories founded on its determination. One may conclude, nevertheless, that it is not improbable that the muscle of the heart is more or less affected in any inflammation of either the endocardium or the pericardium, and that it may even be attacked in rheumatism without signs of implication of either. For the immediate prognosis the extent of the affection of the muscle-walls is all important, and probably also for the ultimate prognosis, since the less diseased the muscle the more perfect the compensation for valvular lesions is likely to be. The general symptoms, the character of the heart action and of the heart sounds, as well as the extent of the dilatation of the heart, may give us some information as to how far the muscle has been involved, but this is difficult to accurately determine, and we are often compelled to await the issue rather than to predict it.

It is a matter, of course, of vital importance to be able to distinguish the first signs of involvement of the heart, and authors differ somewhat considerably in their descriptions. My own experience makes me agree with those who maintain that the most important points are an alteration in the heart action and an alteration in the heart sounds. The heart action becomes excited, the first sound blurred, and later replaced by a murmur which tends very early to be loudest at the apex; the second sound, too, may be reduplicated quite in the early stages. Where there already exists valvular disease it is more difficult to diagnose a fresh attack of endocarditis, and we have to depend upon frequent examinations of the heart to help us. The supervention of pericarditis, as shown by definite friction, clears away any doubt as to the existence of cardiac inflammation, as does also the signs of implication of a fresh valve. A single attack of endocarditis, even when associated with pericarditis and myocarditis, does not necessarily permanently damage the heart. It is in the experience of all of us that after such an attack of rheumatic inflammation of the heart the resulting murmur and dilatation may disappear, leaving the heart perfectly competent, as is shown by the subsequent history of the patient. One has to be extremely cautious, however, in giving a prognosis where a murmur has once existed, since we sometimes find after an attack of rheumatism that the newly developed mitral murmur disappears completely, only to subsequently re-appear and to stay permanently with the contraction of the mitral cusps and their chordæ tendineæ. Even in some cases where no murmur or other sign of cardiac inflammation has been observed we find subsequently that permanent damage has been done to the heart. It is necessary, therefore, after an attack of rheumatism, to examine the patient's heart at intervals during a period of many months before deciding whether there is or is not a permanent valvular lesion. According to some observers complete recovery from a rheumatic inflammation of the heart is more frequent in children. However that may be, an attack of rheumatism in a young child often does not remain the only one, but is commonly followed by a second attack, and this possibly by many more. Such repeated attacks of rheumatism are, of course, well known in adults, but the liability to them is much greater in children, and is greater the younger the child. With each repetition there is a liability for the heart to be affected, and it is possible that this liability increases with each succeeding attack. Thus there is but little chance for the heart escaping in a child who suffers from such repeated attacks of rheumatism.

Cardiac inflammation is the most important event in an attack of rheumatism, and its frequency is much greater in children than in adults. Arthritis, which forms such a marked feature of adult rheumatism that the laity and even, to some extent, the profession are prone to apply the adjective rheumatic to all obscure joint affections, is a much less conspicuous feature in children. Vague and fleeting joint pains are, however, common, and we may state as a maxim that the more definite the arthritis the more likely the heart is to be affected. Of course, all unexplainable joint pains in children

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should be regarded with great suspicion, including "growing pains," and the parents should be warned to take special care of the little patients.

Rheumatism in children shows itself in many other ways besides arthritis, and joint pain is sometimes almost or completely absent. One of the most common of these ways is an attack of tonsillitis. Unfortunately, there is nothing distinctive about a rheumatic tonsillitis, so that we are unable to make a diagnosis, unless there are or have been some other more definite signs of rheumatism. The remembrance, however, that tonsillitis may be rheumatic, and that it may even be sometimes the first manifestation of rheumatism, should make us enjoin care in the management of the patient, and ourselves guarded in our prognosis.

A stiff-neck, too, in a child is very frequently rheumatic in its origin, and may or may not be associated with other signs of rheumatism.

Various skin affections have been described in association with rheumatism, and are more common in children; of these the most frequent are various forms of erythema. The erythematous eruptions are generally associated with other signs of rheumatism, and occur chiefly in the more severe cases; hence they are of bad prognosis. Hæmorrhage sometimes takes place into the erythematous patches, and is occasionally so considerable as to deserve the name of purpura rheumatica. There is one particular form of erythema which, while it occurs independently of rheumatism, is commoner in rheumatic patients, and is sometimes associated with endocarditis without other signs of rheumatism. This erythema nodosum, as it is called, is accompanied by fever and has a more or less definite course. It may be likened in this respect, and in its tendency to occur in rheumatic patients, to tonsillitis.

One of the most remarkable features of rheumatism in a child is its frequent association with emotional disturbances. The rheumatic child is essentially a nervous child, especially if it be of the female sex, is often easily frightened, subject to violent and causeless attacks of weeping, and incapable of application. This want of emotional balance may appear before the first attack of rheumatism, or may only develop after an attack. Anyway, it is not uncommonly exaggerated during an attack. One is at once struck by the similarity of this want of emotional control to the same want of control that is such a conspicuous feature of chorea. Chorea, like rheumatism, is very uncommon before the age of four, and both are at their maximum frequency between the ages of six and twelve. The frequent association, too, of the two diseases in the same patient cannot be questioned, and an attack of one disease often immediately precedes that of the other, or they may occur simultaneously. In many cases the emotional disturbance so frequent in rheumatism is further accompanied by an exaggerated fidgetiness, so that it is difficult to be quite sure whether the child is suffering from slight chorea or not, and between this excessive fidgetiness and the most severe chorea every gradation may be found. In this connection we may remember that the most luminous definition of chorea is that of Dr. Sturges, viz. "an exaggerated fidgetiness." Chorea, then, is one of the manifestations of rheumatism in childhood, and a common manifestation, but this is a very different thing to saying that chorea is rheumatism or only caused by rheumatism; tonsillitis is a manifestation of rheumatism, but is by no means always rheumatic in its origin. If we look at this question from the opposite point of view, viz. from the chorea side, we find that a very large proportion of cases of chorea never show any sign of rheumatism whatever. In 112 consecutive cases of chorea seen by me at the East London Hospital for Children, thirty-nine had definite signs of rheumatism, which shows a rather higher proportion of rheumatic patients than the 30 per cent. given by most writers; some observers, however, put the proportion as high as 50 per cent. or more. In these collections of cases evidence of recent or old endocarditis is regarded as evidence of rheumatism, but it must be remembered that endocarditis may occur in chorea without any other evidence of rheumatism, and it is by no means proved, although it is very probable, that such endocarditis is necessarily rheumatic. Too large a proportion of cases of chorea at any rate are unassociated with rheumatism for us to be justified in declaring that chorea is always a manifestation of rheumatism.

Perhaps the most important of the outward manifestations of rheumatism in children are the subcutaneous nodules which are prone to appear over bony prominences and over the tendons at the wrists and ankles. About a quarter of the

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cases of rheumatism with definite arthritis occurring before the age of twelve years show these nodules. They are much less frequent in adults, but may be found fairly often if carefully looked for. The finding of these subcutaneous nodules in a patient is of value to the physician in two ways. First, they are an absolutely certain sign of rheumatism, and clear up any doubt we may have as to whether the patient has had rheumatism or not. It is true that somewhat similar nodules do occur under other circumstances. In the chronic osteo-arthritis of adults, for instance, as in a recent case of my own, subcutaneous nodules sometimes appear over the olecranon, and nodules are common near this situation in rheumatism. These nodules are exactly similar in appearance and feel to ordinary rheumatic nodules, and apparently have the same histological structure, but their clinical history is different, since they last unaltered for a very long time, even many years, while a rheumatic nodule has individually a comparatively short existence, measured by weeks or months, practically never lasting more than a year. We do, indeed, find that a rheumatic patient may exhibit nodules over several years, but then the nodules come out in successive crops and fade away after a short existence.

The second way in which the occurrence of nodules is of practical help to the physician is with regard to prognosis. It has been found that the patients in whom an eruption of nodules takes place almost invariably have decided organic valvular disease of the heart, or very soon develop valvular disease. Very small nodules have with extreme rarity been found in patients whose hearts were unaffected; I have myself seen one such case—a child with chorea. With these very rare exceptions the rule holds that the presence of nodules implies organic disease of the heart. In a rheumatic child the ultimate prognosis depends upon whether permanent damage has been inflicted on the heart or no. Further, the presence of nodules implies that the rheumatic poison is still active, capable at any moment of exciting fresh cardiac inflammation.

Rheumatic nodules are, as has been previously pointed out, subcutaneous, and are movable over the subjacent bone. Over the scalp and the patella, however, they tend to adhere to the bone. Occasionally a swelling develops in connection with the periosteum, a true periosteal rheumatic node, the favourite situation for which is over the shaft of the ulna near the elbow, but rheumatic nodes are rare, and are always associated with numerous nodules. The number and size of the nodules are also of prognostic importance, since the more numerous and the larger the nodules the more severely, as a rule, is the heart affected, but even apart from the severity of heart affection clinical experience shows that large and numerous nodules are of the gravest import.

In the treatment of rheumatism in the child we have rather different problems to face than in the adult. Only rarely have we urgent pain and high fever to relieve. What is required of us is to prevent, if possible, cardiac inflammation, to prevent the valves being permanently damaged should the heart, unfortunately, be involved, and to prevent a recurrence of the rheumatism. In this connection we must remember—(1) that rheumatism in children is specially prone to recur; (2) that it is specially prone to involve the heart, and that this liability is increased with each successive attack; (3) that a complete recovery after an attack of cardiac inflammation is, perhaps, more frequent in the child than in the adult; (4) that we are better able to prevent a patient from being attacked by rheumatism than we are able to prevent the heart being affected after a rheumatic attack has commenced.

In the prevention of a recrudescence of rheumatism personal hygiene plays a very important part. It seems quite certain that exposure to damp and the living in damp localities may bring on an attack of rheumatism in susceptible persons. One would therefore endorse the usual advice to such persons to avoid, if possible, living in low-lying damp places, to be careful not to get their clothes wet, and not to remain in wet clothes. They should also wear woollen garments, especially next the skin.

The use of drugs in the cure and prevention of rheumatism is a more debatable question. There can be no doubt that salicin and the salicylates alleviate the symptoms of rheumatism in the adult, bringing down the temperature and relieving the pain, but it has been argued that they do not cure the disease, and that they are harmful in themselves. Any unpleasant result of the administration of the salicylates seems to me to be uncommon

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with proper doses, and to be generally unimportant since they cease directly the dose is reduced or the administration stopped. In my opinion, too, sufficient evidence has been accumulated that the continued administration of the salicylates has some influence in preventing the occurrence of relapses. In spite of the fact, then, that certain of the manifestations of rheumatism are unaffected by the salicylates, one would recommend as a routine treatment that they should be given frequently, every three to four hours, in full doses (gr. ¾ for every year of the child's age) until the active signs of rheumatism disappear, and then that similar doses should be given three times a day for one or two weeks. During the attack the patient should of course be kept in bed, clad in flannel, and should have light diet, chiefly milk. After an attack the return to a more generous diet should not be too early, since there is a general consensus of opinion that such an early return tends to cause a relapse. Considerable anæmia is almost invariable after an attack of rheumatism, and requires treatment with iron.

It is doubtful whether we have any means of preventing cardiac inflammation after a patient has been attacked by rheumatism. It is possible, however, that the longer the other symptoms continue the more likely the heart is to be affected. The administration of the salicylates by cutting short an attack may possibly in some cases prevent endocarditis. Thus in the main the prevention of cardiac inflammation depends upon the prevention of the rheumatism.

When the heart has already been involved our main object should be to give it a chance of recovery. There seems to be no drug that has any influence over the course of the cardiac inflammation. Since, however, with the continuance of active rheumatism fresh cardiac infection may occur, it is necessary to continue the administration of the salicylates. The salicylates have been found to have a depressant action on the heart, but the continued action of the rheumatic poison, and the possibility of fresh cardiac infection, are of more importance than this depressant action of the drug. Hence the drug should be persisted in, unless there are urgent signs of cardiac failure. Our main hope, however, in promoting recovery from cardiac inflammation must lie in giving the heart as little work as possible. In the horizontal position the heart has less to do, and beats decidedly less frequently than in the erect position. The patient, then, should lie supine in bed and remain so for a lengthy period after the subsidence of the rheumatic attack, *e.g.* at least a month. In case of restlessness and excited action of the heart, a small dose of opium may be given, which is our best means for quieting the heart and the nervous system of the patient. This rest treatment should be carried out not only where there have been definite signs of cardiac inflammation, but even after any marked attack of rheumatism in a child, since it is impossible to be sure that there has been no endocarditis during the attack. Unfortunately this is a counsel of perfection, as both the child and its relatives generally refuse to carry out the treatment after the child feels nearly or quite well. Such an enforced rest may be conjoined with the treatment recommended by Dr. Caton, which, in his hands, has given excellent results. This consists in giving sodium iodide in appropriate doses, as an alternative, and applying small blisters repeatedly in the lines between the middle of the clavicles and the nipples on either side.

Where permanent damage has been done to the valves of the heart, it is still of the greatest importance to prevent further attacks of rheumatism, each of which might be accompanied by fresh cardiac inflammation. Valvular heart disease in children is only too often associated with frequent and persistent eruptions of nodules, an evidence of continued rheumatism. It is obvious, however, that we cannot keep these children in bed all the time that they show signs of rheumatism in the form of nodules, since these may last many months, and in successive crops many years. Salicylates, too, seem to have no influence over the appearance and continuance of rheumatic nodules. Hence in cases of chronic valvular disease of the heart in children our treatment, beyond keeping the patient as far as possible from exposure to those conditions which tend to bring on a rheumatic attack, has mainly to be directed towards the maintenance of compensation in the damaged heart.

SHELMIRE ('American Medicine,' June 7th) reports a case of dermatitis medicamentosa in the form of an unusually extensive and severe erythema produced by the ingestion of mercury in the form of calomel.—*Philadelphia Med. Journ.*, June 21st.

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THE SURGICAL TREATMENT OF FIBRO-MYOMATA OF THE UTERUS, WITH ILLUSTRATIVE CASES.*

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GENTLEMEN,—I propose to briefly review the surgical treatment of myomata, illustrating my remarks with specimens obtained from cases on which I have operated at the Tottenham Hospital during the time I have been connected with this institution. All the cases met with are here included, numbering twenty in all.

It would be beyond the scope of this paper to discuss the pathology and diagnosis of these tumours, and the indications for surgical intervention. On the last point only will I summarise my views, which coincide, as I believe, with those of the majority of gynæcologists at the present time.

Fibro-myomata which give rise to no symptoms should not be interfered with. The conditions which call for surgical treatment are the following:

1. Excessive hæmorrhage.
2. Pressure symptoms.
3. Great and continued pain.
4. Chronic disablement due to one or more of these conditions.
5. Rapid growth.
6. Evidence of septic or degenerative changes.
7. Interference with labour.

The operative measures which may be required may be classified as follows:

(*a*) VAGINAL OPERATIONS.
1. Vaginal myomectomy.
2. Vaginal enucleation.
3. Vaginal myomectomy or enucleation with morcellement.
4. Vaginal hysterectomy.
5. Vaginal hysterectomy with morcellement.

(*b*) ABDOMINAL OPERATIONS.
1. Abdominal myomectomy.
2. Abdominal enucleation.
3. Abdominal hysterotomy.
4. Abdominal hysterectomy.
5. Abdominal panhysterectomy.
6. Cæsarean hysterectomy.

I shall consider these procedures briefly in their order.

(*a*) Vaginal Operations.

1. VAGINAL MYOMECTOMY.—By this is meant the removal entire of a pedunculated (polypoid) myoma from the interior of the uterus. The tumour may project into the vagina, or it may be confined within the uterine cavity, when the cervical canal will require to be dilated in order to

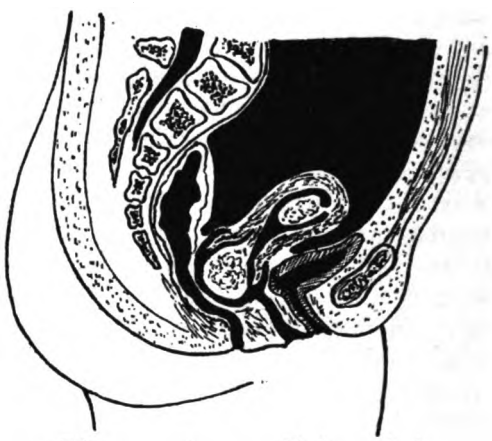

FIG. 1.—Fibro-myomata removable by vaginal myomectomy.

remove it. This method is limited by the size of the tumour; but the limit of size is reached sooner with intra-uterine tumours than with those that have become extruded into the vagina. The types of myomata suitable for this treatment are shown in Fig. 1.

The cases treated by vaginal myomectomy were as follows:—CASE 1.—E. M—, æt. 42 years, married, was admitted on October 15th, 1901. She had had seven children, the youngest being two years old. For two years menstruation had been excessive and frequent, coming on every fortnight. Two weeks ago hæmorrhage became incessant; it came away in gushes, accompanied by many clots. On admission the patient was very blanched and anæmic. Vaginal examination showed that a fibroid polypus the size of a walnut was projecting

* Remarks made, with demonstration of specimens, before the North-East London Clinical Society, Tottenham Hospital, on May 1st, 1902.

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from the cervix. The cervix was large, firm, and everted, the os being the size of a shilling, and the cervical canal occupied by the pedicle of the polypus. She was losing blood freely. On October 17th the polypus was seized with a volsella and twisted round till the pedicle gave way. There was no bleeding from the site of attachment, which was touched with iodised phenol. Convalescence was good, and the patient went home well on the fifth day.

CASE 2.—J. J—, æt. 47, married, was admitted on April 12th, 1902. She had had five children, the youngest being fifteen years of age. Menstruation had always been rather abundant. In January of this year it was very profuse, the blood pouring out, accompanied by clots. In February the same thing occurred, and again the week before her admission. On admission the patient was very anæmic; the cervix was found dilated to the size of a half-crown, and through it projected a fibroid polypus, the size of an egg-plum, with a thick pedicle which filled the cervical canal. The uterus was enlarged, the sound passing in five inches. On April 17th the fibroid was seized with a volsella and rotated till the pedicle was twisted off. It was found to spring from the anterior wall of the uterus, just within the internal os. There was no hæmorrhage, and the cervix was packed with gauze. Convalescence was normal, and the patient went home on the fifth day.

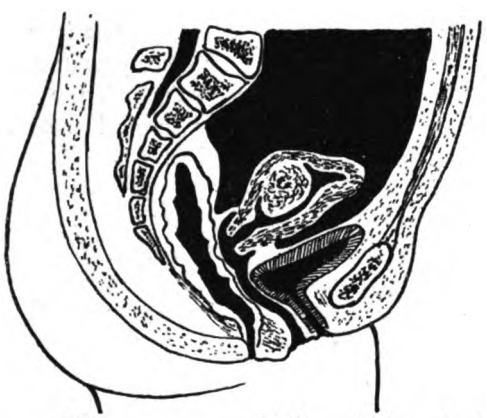

FIG. 2.—Fibro-myoma removable by vaginal enucleation.

2. VAGINAL ENUCLEATION.—This means the removal, *per vaginam*, of a myoma embedded in the substance of the uterine wall. It is always necessary to dilate the cervical canal; and, as in the case of an intra-uterine polypus, it may be necessary to obtain more room for manipulations either by bilateral division of the cervix, or by reflecting the bladder from the front of the uterus, as in the preliminary stage of vaginal hysterectomy, and dividing the anterior wall of the cervix and uterus in the middle line for two to three inches. When the tumour is reached its capsule is incised, and the growth is enucleated.

This type is shown in Fig. 2, and is illustrated by the following case (CASE 3). J. P—, æt. 38, married, was admitted on June 9th, 1900. She had had one child fourteen years previously. Menstruation had always been free. For the last twelve months it had been more profuse, and she had had hæmorrhage irregularly. She had pain in the lower abdomen, aggravated during menstruation. On admission the patient was a rather fat and very anæmic woman; a systolic blowing murmur could be heard at the apex and over the aortic and pulmonary areas. The uterus was enlarged, its body being the size of an orange. The cervix was normal. On June 12th the cervix was dilated and incised down each lateral margin. A small myoma with a broad base was found attached to the posterior wall of the uterus. This was enucleated, as was also another small myoma in the anterior wall. Convalescence was normal.

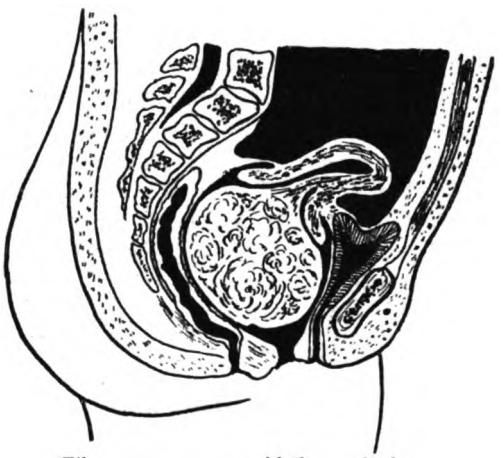

FIG. 3.—Fibro-myoma removable by vaginal myomectomy with morcellement.

3. VAGINAL MYOMECTOMY OR ENUCLEATION WITH MORCELLEMENT.—This means the removal of a polypoid or interstitial myoma *per vaginam*, after division of the tumour into two or more portions, its size being too large to admit of its removal entire. No hard and fast lines as to size

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can be laid down, because much will depend on the room afforded in the pelvis for manipulation; but broadly it may be said that an intra-uterine tumour of the size of an orange, or a tumour the size of a closed fist lying in the vagina, will require to be divided before removal. With a large intra-uterine tumour, as in Fig. 6, I prefer the operation of abdominal hysterotomy, which will be described later on. Vaginal myomectomy with morcellement was done in the two following cases, which were both instances of tumour lying in the vagina, one being the size of a fist, and the other that of a fœtal head (Fig. 3).

Case 4.—F. L. T—, æt. 43, married, was admitted into the Tottenham Hospital on March 18th, 1900. She had had nine children, the youngest six years old. Menstruation was regular till twelve months before admission, when the periods appeared every fortnight, and she lost much blood, with clots. For three weeks she had had almost continuous hæmorrhage. On the day before admission she had retention of urine for the first time; a catheter was passed, and a large quantity of urine was drawn off. She had had a discharge for six weeks, and bearing-down pains. On admission the bladder was full; twenty-one ounces of urine were drawn off by catheter. Examination of the abdomen showed a tumour with uneven surface, hard, and extending to within an inch of the umbilicus. The vagina was occupied by a large, smooth, rounded swelling coming down to within two inches of the vulva; it felt fleshy, and the finger could be passed all round it. It was a pedunculated myoma, protruding from the cervix; the body of the uterus formed the summit of the tumour.

On March 20th the tumour was removed by the vagina; its size necessitated its division in halves before the pedicle could be reached and cut through. Convalescence was uninterrupted.

Case 5.—E. M—, æt. 38, married, was admitted on May 22nd, 1901. She had been married fifteen years, and had one child, fourteen years old. For the last three years she had suffered from excessive losses, which had much weakened her. She used to pass clots, and once she had an attack of syncope from the loss of blood. She complained of bearing-down pain in the abdomen. On admission the vagina was completely occupied by a large smooth mass. The finger could not reach above it to the cervix in any direction. In the hypogastrium a smaller, hard mass could be felt, resembling the body of the uterus. It having been decided that the tumour was outside the cervix, it was removed by vaginal morcellement. When the greater part had been removed, the pedicle was found to spring from the posterior part of the cervical canal, the uterine cavity not being encroached upon. The cervix itself was spread out over the upper portion of the tumour, like an inverted cone, three inches in diameter. The cervix was packed with gauze. The mass weighed altogether one and three quarter pounds. Convalescence was uneventful, and the patient went home on the fourteenth day.

4. Vaginal Hysterectomy.—By this is meant the complete removal of the uterus by the vaginal route. It is commonly performed on account of cancer; but its application to myomata is exceedingly restricted, because very few myomatous uteri small enough to be removed through the vagina require to be dealt with by surgical means. I have not had a case of this kind.

5. Vaginal Hysterectomy with Morcellement.—By this is meant the removal of the uterus through the vagina after its division into two or more portions, when it is too large to be removed entire. This plan has its advocates; but in my opinion abdominal hysterectomy is, under these circumstances, a simpler and safer operation, and I have no cases to illustrate the method.

(*To be concluded.*)

Changes in the Pulse in Childhood.—G. A. Dotti ('Rivista Critica di Clinica Medica,' May, 1902) asserts that the pulse undergoes greater changes normally and pathologically in children than in adults. In typhoid fever arrhythmia appears from the fifteenth to the thirtieth day, and disappears from the thirty-fifth to the fiftieth. In pneumonia the pulse resembles that of typhoid fever more than it does in other pulmonary diseases, in which there is irregularity rather than a true intermittence. In influenza the arrhythmia appears somewhat early. In diphtheritic angina of slight import there is merely tachycardia and a small pulse, but in grave cases there is bradycardia. This also occurs in the convalescence from scarlatina.—*Medical Record*, June 21st.

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* *Specially reported for The Clinical Journal. Revised by the Author.*

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A CLINICAL LECTURE

ON

CYSTS OF THE NECK.

Delivered at St. Bartholomew's Hospital, March 17th, 1902.

By ANTHONY BOWLBY, C.M.G., F.R.C.S.,

Assistant Surgeon to the Hospital; Joint Lecturer on Surgery and Lecturer on Diseases of the Throat.

GENTLEMEN,—I propose to lecture to you to-day upon cysts of the neck. Of course, it is impossible in one lecture to do justice to the whole of that subject. First of all, let me say that with regard to cysts in connection with the thyroid gland I shall have nothing to say in this lecture, except to remind you at the beginning that thyroid cysts may appear in parts of the neck where you might not expect to find them, because portions of thyroidal tissue may be as if it were out of place in different parts of the neck. You have to remember that portions of the thyroid gland, represented by what is called the pyramid of the gland, may become detached, and that consequently thyroid cysts may appear quite high up in the neck. They may be either mesial or lateral. About thyroid cysts I have nothing particular to say to-day, but I hope to have an opportunity of giving you a separate lecture upon diseases of the thyroid gland. Putting thyroid cysts aside, you may divide cysts in the neck into two large classes: those which are lateral and those which are median.

First of all, let us consider those which are laterally placed. Let me remind you in the first place of the development of the neck, because that has much to do with the cysts which we shall speak about. In connection with the development of the neck there are four branchial arches or clefts, and the remains of these clefts may be left in the neck, so that although normally there ought

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to be a complete closure of what was once an opening into the pharynx and an opening into the skin, occasionally there is left a communication in the skin which passes down into a tube of tissue in the neck. Under these circumstances you have to do with what is called a "branchial fistula." In many cases that is the key of the explanation as to how you get certain cystic diseases in the neck. The most common situation for this branchial fistula is low down in the neck, generally in front of the border of the sterno-mastoid and at about the level of the cricoid cartilage; and in this situation there may be a tiny aperture, no larger than will admit a bristle or pin, and sometimes will not admit even the finest probe. From this there may ooze a certain quantity of clear fluid, which is increased during mastication. That is a branchial fistula, and at the orifice of this fistula there is not infrequently found a small portion of cartilage, to which the name of an "accessory auricle" is given. An accessory auricle may be formed anywhere in the neck where a branchial fistula exists. In the next place, I will show you these drawings, which illustrate accessory auricles. In this picture of a girl there is, low down in the neck, one of these accessory auricles, marking the situation of a branchial fistula. If you open the fistula and track it, you will find it opens into a tube much larger than the orifice would give you any impression of, and that this tube passes upwards and inwards until the end of it is found attached to the pharyngeal wall. It generally lies superficial to the vessels. This is a tube connected with a branchial fistula which I removed from the neck of a boy in the hospital. The history of the case was that the boy was troubled with the oozing of clear fluid from his neck. It was very difficult to find the tiny aperture through which this came, and afterwards, recognising that one had to do with a branchial fistula, I set to work to remove the whole of the tube by making an incision in the neck and following this tube along until it came to the end in the pharyngeal wall. Here is the tube in the bottle, two or three inches long. When you examine a microscopical section of it you find that it is composed of a lining membrane of epithelium, and that around that is placed some fibrous tissue and some lymphoid tissue, just like lymphatic-gland tissue. Now, supposing for a moment that there is in the neck that tube of tissue which I have shown you here, but that there is no opening on the skin and no opening into the pharynx, and supposing that this tube of tissue secretes clear fluid, it will become distended by that fluid, and will, of course, in that way, gradually become a cyst. That is how these lateral dermoids of the neck are formed in connection with the remains of branchial clefts, the openings of which have been completely closed. And there are two common places for these cysts to be found; one is close underneath the maxilla, and the other is over the anterior triangle of the neck, extending in the same direction as that followed by this tube of tissue which I have indicated. Here is a drawing of a branchial cyst or dermoid cyst in the submaxillary region. I have not got a specimen to show you of that, but I can tell you of cases where they occurred in the anterior triangle of the neck at the level of the thyroid and cricoid cartilages.

Next, at what age and under what circumstances and clinical conditions do they occur? Though these, of course, are congenital in origin, they seldom make their appearance until, at any rate, puberty, and sometimes not until quite late in life. Therefore you are liable to be mistaken if you think that, because they are congenital in their origin, they necessarily form tumours in early life. I removed a large branchial cyst or dermoid cyst from a gentleman æt. 54. There was no doubt that it was congenital in origin, but there had been no filling up of this potential cavity until that age.

Next, as to the clinical characters of these formations. They present themselves as smooth, elastic, and not very tense tumours. They are definitely fluctuating; they cause no pain; and until by their size they attract attention, they are not generally suspected at all, and as they grow slowly they often attain a considerable size before the patient notices them. They only gradually displace the structures in the neighbourhood and stretch the skin. And so it happens in many cases that when you first see these swellings they are already the size of a pigeon's egg, or may be even the size of a hen's egg. They are often mistaken for chronic abscesses. About three months ago a patient was sent into the hospital, in Stanley Ward, with a swelling of the neck, and with a history that about a year previously she had had an abscess in her neck. This was cut into and some fluid evacuated. It afterwards healed. The

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fluid re-accumulated and burst its own way out the second time at the place where the incision had been made. Again it practically healed, but left a slightly discharging place. This became septic, and consequently the girl's neck became inflamed. It was opened up and a tube put into it. Again after this it became contracted, again became septic, was again distended, and an abscess formed. This was a dermoid cyst in the neck, which had been mistaken for an abscess and opened as one, and had been treated for more than a year in different ways by the evacuation of this fluid and by the sealing up of the sac, yet, again and again this fluid had re-collected. The treatment was to remove the cyst, and when it had been completely removed it was found that it lay close to the pharyngeal wall, that it lay upon the vessels, and that the spinal accessory nerve passed immediately beneath it to reach the sterno-mastoid muscle. That is the characteristic position of these sub-maxillary dermoids. It is easy to remove these tumours if you do not mistake them for abscesses and make them inflame. There was a great deal of trouble experienced in getting rid of this tumour in the neck of this girl. It had been frequently inflamed, and was closely adherent to the surrounding structures, and therefore its removal was a matter of a good deal of time and trouble. If you recognise them early and operate upon them when they are not inflamed their removal is a comparatively simple matter. That is one form of laterally placed cyst in the neck of congenital origin.

The next form of laterally placed cyst in the neck of congenital origin is altogether different in its etiology, namely cystic hygroma of the neck. It is called also by many other names. This is most often situated in the posterior triangle of the neck, and it is composed of either a single thin-walled cyst or several thin-walled cysts; or of cysts with a certain amount of soft yet tough fibrous tissue connecting them; or it may be mostly composed of soft fibrous tissue and very little cyst. These are mesoblastic tumours. They have nothing to do with the enclosure of epithelium; they have nothing to do with branchial clefts. They are formed from the mesoblastic tissues out of which the muscles and the other connective tissue of the neck are made. And in the process of differentiation of mesoblast to form the different structures in the neck a certain amount of this tissue remains, as it were, insufficiently differentiated, and these conditions result. These tumours, congenital like the others, are more truly congenital than the branchial cysts, because they are present at birth; they are often already distended at birth. Consequently you find them not in young adults or in people who may be in middle age, but in infants. Now, clinically they may be very small, or they may be as large as a child's head. Here is one which was allowed to last for a long time, and which you see forms a great swelling in the posterior triangle of the neck, extending from the mastoid process to the clavicle. That is a very large one. These cystic hygromata of the neck, if left alone, occasionally spontaneously disappear. That is a curious fact about them. But more often they remain and slowly grow, but their growth is not in proportion to that of the child, so that an abnormality which seems big for an infant does not appear relatively large for an older person, These cysts present themselves either as smooth, elastic, or fluctuating tumours, and there may be either one cavity and one collection of fluid, or they may be loculated in proportion as there are numbers of cysts, and in proportion as there is firm fibrous tissue in them. In some cases they are irregular and lumpy, and they are always deeply placed, and always more or less fixed to the deeper muscles of the neck amongst which they lie. As a complication, occasionally there may be hæmorrhage in them. A little time ago a patient was in this hospital who had one of these cysts, and when it was tapped it was found to contain blood. The specimen from which this picture was taken also contained blood. So if you find a cyst in the neck containing blood, in a little child, it is probably a cystic hygroma into which bleeding has taken place.

Now with regard to treatment. They are ordinarily best treated by either simple tapping, which occasionally cures the single large cysts, or by tapping and the injection of iodine in the form of Morton's fluid; or it may be necessary to do complete resection. But in proportion as these tumours are composed of solid material, and in proportion as they pass very deeply into different portions of the neck, or behind the clavicle or behind the jaw, so as to enter into inaccessible regions, they are best left alone. And thus it comes to pass that some of these cases are not

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suitable for operation at all, for if you operate upon them by tapping or injection when they are deeply placed, the inflammation and swelling which may ensue may endanger the life of the child. So that where there is a single cyst, and it is in an accessible region, it may be a simple matter; but in propoportion as there are many cysts and much fibrous tissue, and they grow from inaccessible parts from which they cannot be extirpated, it becomes safer to leave them alone.

Dealing still with lateral cysts, here is the picture of a man with a large cystic swelling in his neck. We have no very good history of that case except that the man was aged forty-five and had noticed a swelling in his neck for three years. At first it was about the size of a marble. Two drachms of viscid fluid were drawn from it. It went down considerably, but rapidly increased in size again, and the skin over it was ulcerated. There is no doubt it was a case of malignant disease of the neck. These cysts in connection with malignant disease of the neck fortunately are rare.

A man æt. 48 was admitted into the hospital with a swelling in the right side of the neck, just like a chronic abscess. The history was that it had been there for a few months; it was about the size of a small cocoanut; it was tense, and the skin over it was rather œdematous; it was painful; quite evidently there was fluid. My house surgeon made an incision into this swelling, opened it, and let out a considerable quantity of blood-stained fluid, which was rather thick, but contained no pus. On putting his finger into the opening which he had made he found the inside of the cyst was rather rough and hard, and he found that the cyst had a base which was very firm, indurated, and deeply attached to the neighbouring muscles. That cyst continued to discharge blood-stained fluid, and very soon there came to the surface a growth of epitheliomatous nature. From extension of this disease in the neck he died. After his death we found this cystic epithelioma of the neck. You see the large cavity which contained fluid, but in itself now it is a mass of epithelioma, broken down in the middle and infiltrating the neighbouring parts. This is probably one of those cystic formations which in all probability begin as an epithelioma of an unobliterated branchial cleft. I showed you just now a branchial fistula leading into a small tube of tissue, lined by epithelium, with ordinary squamous epithelium implanted in the midst of lymphoid tissue. That epithelium, just like any other epithelium, may be the seat of epitheliomatous growth; and these cysts of the neck, which are very deceptive, and which simulate abscess, pour their fluid, which results from the breaking down of the epitheliomatous material, into the little cavity, so that there is a cyst in that position, but lined by an epithelioma, and not by ordinary epithelium. In this patient, whose case I have narrated, there was no other epithelioma anywhere to which this might have been secondary.

The next thing I have to remind you of is that a certain number of swellings in the neck which are apparently cystic are epitheliomatous glands breaking down. For instance, a man has epithelioma of his tongue, or of his lip, or of the floor of his mouth, which although it may be small, may nevertheless cause the appearance of very large glands in his neck. And it is quite possible for the original epithelioma to be lost sight of, and for the patient to consult you merely about the swelling in his neck, which swelling may have the appearance of suppuration. It is an elastic tumour with discoloured skin adherent to it, and the fluid is puriform. So you might think you had to do with epithelioma of the neck. In reality it is due to primary malignant disease elsewhere. Lastly, let me remind you that some laterally placed swellings which simulate cysts are the chronic abscesses of tuberculous disease. These are not to be included in the "cystic swellings" of the neck.

Next we will consider "mesial cysts," that is to say, those placed in the middle line. Beginning from above, you may first of all find them in the uppermost part of the neck, protruding both in the neck and in the mouth. So when you look inside the patient's mouth you may find the swelling of a cyst, which, when he shuts his mouth, is found to bulge beneath the jaw. That is the highest placed mesial cyst of the neck, a supra-hyoid or submental cyst. It is commonly a dermoid. Between the two halves of the tongue there is a process of epithelium, which may be shut in, the skin closing over it on the one hand and the mucous membrane on the other; and there may thus be subsequently developed between the two halves of the tongue a dermoid cyst. This may bulge into

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the mouth and lift up the floor of the mouth, and lift up the tongue towards the roof of the mouth; or, on the other hand, it will bulge between the hyoid bone and the chin, and form a swelling which is more or less obvious in the neck itself. Just as in the case of dermoids in the side of the neck, so in these cases you may never see these cysts in children. They are not there at the time of birth—they take time to fill up—because the cyst is filled with the secretion of the glands which lie in its walls, and they pour their contents into its closed cavity, and thus a cyst is formed. This takes time, and consequently these cysts are not found until early adult life. They are most commonly seen between the ages of fifteen and twenty-five. Most of the cases I have seen have been in young men; one was in a young woman of about twenty. These tumours are very often mistaken for abscesses which can be emptied, and they are so definitely elastic that they are often punctured. In a great many cases the same trouble arises as when you puncture these laterally placed dermoids, that is to say, suppuration. I saw in one young fellow a troublesome condition. He had had a cyst punctured from inside his mouth, and from having been opened in that way it had become septic, and from a certain amount of suppuration going on always the cyst became fixed in a way which made it very awkward to remove. In Africa a soldier was sent to me from the front because of an inflammation of the mouth and high temperature. He had suppuration in the floor of his mouth. The tongue was lifted up, so that he could hardly move it or speak, and he could not masticate. This was a case where a dermoid cyst of the mouth had been opened a long time previously and had become septic, and the inflammation had spread in the floor of the mouth and caused all this œdema and difficulty of moving his tongue, so that the swelling threatened his life. It is of the greatest importance to recognise all these dermoid cysts in the neck, and not to open them under the impression that they are chronic abscesses. Each of them feels so much like a chronic abscess that, unless you know what it is likely to be, you are liable to open it under the impression that it is an abscess.

With regard to treatment, if you recognise such a cyst you can open it by an incision in the middle line of the chin down to the hyoid bone, and by holding the muscles apart you can gradually separate the cyst from its surroundings. After having nearly separated it, it is best to puncture it and empty out some of the contents, and then, by passing your finger into the cyst, you can strip it off the remaining tissue, take it out, and leave practically no scar, for a scar between the chin and the hyoid bone is very much out of sight, and this should be a wound which should heal by first intention.

Let us go now to the episternal notch. This is a region where dermoid cysts again are found. A cyst which appears on the uppermost part of the sternum may pass a considerable distance into the anterior mediastinum. A cyst which is placed at the base of the neck may be a part of a much larger cyst which passes into the anterior mediastinum itself, so that cases of this sort have had to be treated by trephining part of the sternum in order to get rid of the contents of the cyst which are pressing upon the intra-thoracic viscera. Between these two places I may remind you of thyroid cysts, but only to put them aside.

But above the level of the thyroid gland, and between it and those upper dermoids in the submental region, you may find cysts containing clear or slightly stained, glairy, sticky fluid. These are either bursal cysts or cysts in connection with the thyro-glossal duct. In deglutition the movements of the thyroid cartilage are facilitated by an interposed bursa, which passes up behind the hyoid bone. This bursa may be distended and may form a cystic swelling in the middle line of the neck, between the thyroid cartilage and the hyoid bone, therefore lower down than the upper-placed dermoids. It is not amongst the muscles because there is not much muscular tissue in that region. It is not large, but it is much more tense and elastic than most cysts are. It is about the size of a hazel-nut; sometimes it may be as large as a walnut. Such a cyst passing up behind the hyoid bone is a source of the greatest trouble to remove. My advice is, if you have such a cyst as that, do not be in a hurry to remove it. Find out how much trouble it causes the patient. Generally that is not a great deal, and then you may treat it by any form of procedure which you find useful for a large bursa anywhere else. You may paint it with iodine, and you may apply such things

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as Scott's ointment. If you think the patient has got anything like a gouty deposit you may treat him for gout. Occasionally you may be driven to operate. You may ask, "Why should not you operate? It is simple enough to remove a bursa." Yes, up to a certain point it is the simplest thing to remove, but with regard to the uppermost part of the cyst passing behind the hyoid bone, all the time you are operating the patient is swallowing, and you cannot stop these movements during anæsthesia. The uppermost part of the bursa often lies behind the hyoid bone in front, and is only separated behind from the inside of the pharynx by the pharyngeal aponeurosis. You do not want to open up the thyro-hyoid space, and you do not want to divide the hyoid bone. Then you are working in a very narrow space, and you will find that the removal of the upper part of this bursa is a very difficult task. Otherwise it would not be the fact that in many cases in which this operation is undertaken the patient is left with an opening in his neck which discharges fluid and will not permanently heal. I remember a patient being brought into this hospital who had had one of these bursal tumours operated upon four times: first of all, for removal of the swelling; secondly, to try to close the opening; thirdly, to try to close the opening; and fourthly, ditto; and finally, when he went out the opening was still there. It fell to my lot to operate the fourth time on another patient. One surgeon had operated upon him twice, then he went to another surgeon, and finally came to me, and I am sorry to say that I did not close it. I removed the remains of some bursa beneath the hyoid bone, and ultimately, though I did not close it at that time, the operation was followed in the long run by closure. But for several years it was liable to break out again if the patient caught cold and it got inflamed. The trouble in such cases is that the sac is at a place where you cannot get at it easily, and after having completed your operation it is hard to keep the parts at rest. There is very little thickness of tissue to bring together, for there is very little subcutaneous tissue between the skin and the thyroid cartilage, and it is a very difficult thing to get these small bursal swellings satisfactorily excised and the wound healed afterwards. It is for that reason I advise you not to be in a hurry to undertake the removal of these tumours, simple as the operation is in one way, because you may leave the man with a fistulous opening in his neck which secretes fluid, and as soon as the patient eats or talks the secretion is increased; and he is put to a great deal of trouble and inconvenience by the opening, which is in a part which is difficult to dress.

One might have said a good deal more about cysts of the neck than I have, but there is no time to go further. As I have said, cysts in connection with the thyroid gland form an altogether separate subject; but I would ask you to remember in connection with mesially placed cysts that cysts of the thyroid gland are at least as common as all the other cysts I have mentioned put together. Therefore in the middle of the neck you have always to keep before you the possibility of any cyst originating from the thyroid gland.

Röntgen Rays in Cranial and Cerebral Affections.—Benedikt calls the attention of the profession to the information to be derived from radiographs of the skull after traumatisms causing the so-called "shock neuroses." He takes radiographs of the profile and also of the front and back of the head, and finds that in many instances the bones are abnormally permeable for the rays at the points where pressure is painful, showing the existence of some inflammatory process at this spot. The facts demonstrated by radiography have been confirmed by the anatomic findings later in many cases in his own and Kienboeck's experience. In cases of pachymeningitis, the inner layer of the bones of the skull may be less permeable than normal. This corresponds to the hæmorrhagic tendency of pachymeningitis. In cases of unilateral meningitic swelling or hydrocephalus externus, the increased permeability will attract attention, while pachymeningitic hæmorrhages are distinguished by the reverse phenomenon. These shades of difference are best seen on the plate, and physicians should learn to read the plates and thus avoid the further errors of the copy. Röntgen rays thus applied avoid all confusion from simulation or aggravation on the part of the subject, and reveal the anatomic basis for traumatic neuroses which is too often denied.—*Journ. A. M. A.*, July 5th, 1902.

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ON

COMPLETE REMOVAL OF THE GALL-BLADDER (CHOLECYSTECTOMY).

By J. BLAND-SUTTON.

In the Bradshaw Lecture, delivered at the Royal College of Surgeons, 1890, Sir Spencer Wells, in reference to the surgery of the gall-bladder, stated that he could "almost foresee that the general rule in future will be to expose the gall-bladder, empty it by a syringe, raise the liver, protect the stomach and intestines by sponges, tie the cystic duct with two ligatures, divide it between them, separate the gall-bladder from the liver by the knife or scissors, and then close the abdominal wound" ('British Medical Journal,' 1890, Vol. ii, p. 1467). This forecast has not been realised. To open an enlarged gall-bladder, evacuate the contents, whether mucus, pus, or biliary concretions, and then secure the artificial opening to the edges of the abdominal incision is in many instances a thoroughly straightforward and fairly safe surgical proceeding. To detach an enlarged gall-bladder from the under surface of the liver, secure its pedicle with silk, and then close the abdominal incision is often a perilous enterprise.

There are cases, however, in which the surgeon is faced with conditions such as follow:

1. The gall-bladder, though containing concretions, is shrunken and so small that its fundus cannot be brought into the abdominal wound.

2. The gall-bladder may be very large and distended with mucoid fluid in consequence of a stone impacted in the cystic duct, yet its walls are so thin as to be translucent.

3. A large, thick-walled, inflamed, and pus-containing gall-bladder, extremely tender, and its cystic duct blocked by a tightly impacted gall-stone.

4. The gall-bladder may be occupied by cancer as well as gall-stones.

In the course of a very large number of operations on the gall-bladder and gall-ducts I have only found it necessary to completely remove the gall-bladder on six occasions. The first patient was a woman æt. 60; she had an enormous ovarian tumour, and, in addition, a fistula under the right costal arch which discharged a small quantity of bile daily. I removed the ovarian tumour, and found it adherent to the under surface of the liver and the gall-bladder. She recovered quickly from the ovariotomy, and some months later I exposed the gall-bladder with its concretions, and, after satisfying myself that the main biliary passages were free from gall-stones, I treated the gall-bladder in the summary fashion suggested by Sir Spencer Wells, and with complete success. I subsequently removed another shrunken gall-bladder containing a few concretions in a similar way, and with success; but when the gall-bladder is much enlarged the operation is by no means simple, as the following case shows:

A woman æt. 28 was placed under my care by Dr. Ferguson, of Norwood, on account of an extremely mobile pyriform tumour, projecting from beneath the right costal arch and reaching below the level of the umbilicus. The tumour was painless except when manipulated, and though free from any traces of jaundice, her health had been indifferent for many months. The tumour was exposed by a free incision in the right linea semilunaris, and proved to be a large thin-walled translucent gall-bladder. The greater part of its anterior surface was covered by an attenuated process of liver. I cut away, by means of two incisions made at right angles to each other, this thin hepatic tissue, and detached the gall-bladder from the under surface of the liver, and then carefully manipulated the calculus impacted in the cystic duct back into the gall-bladder. The cystic artery was secured with forceps and the duct divided. The artery was ligatured with thin silk, and a separate ligature placed upon the stump of the cystic duct. The triangular gap in the free edge of the liver was obliterated by bringing the cut edges into apposition by means of silk sutures; this at once controlled the bleeding. A thin gauze drain was introduced, and the abdominal incision closed by the triple-layer method. The patient recovered rapidly, and returned home on the sixteenth day after the operation, and is at this date in excellent health. The gall-bladder, with the impacted calculus *in situ* and four vagrant calculi in its interior, is represented in Fig. 1. I decided to remove this gall-bladder completely, because these attenuated cysts do not shrink like

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the thick-walled muscular gall-bladder, and are extremely liable, when merely stitched in the upper angle of the abdominal incision, to give rise to a troublesome fistula. I have removed a gall-bladder of this character on two other occasions with equally satisfactory results.

The largest and most formidable pus-containing gall-bladder I have completely removed had a very interesting clinical history, for the patient had been treated by a very able surgeon for appendicitis. In this instance it was necessary to excise a large piece of liver in order to remove the

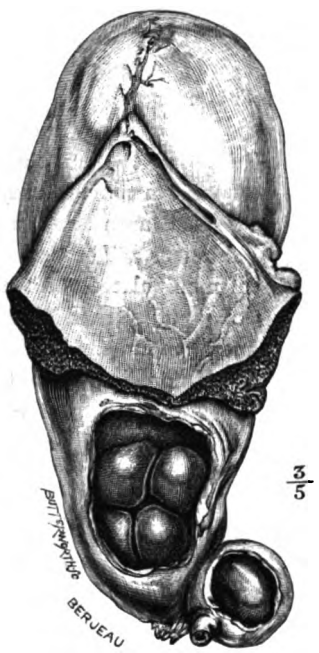

FIG. 1.—A thin-walled gall-bladder greatly distended with mucus secondary to obstruction of the cystic duct by a gall-stone. Removed from a woman æt. 28.

gall-bladder completely. The remote results were very good. (The details were published in the 'Medical Press and Circular,' July 4, 1900, p. 12.)

It is by no means uncommon in dealing surgically with big, inflamed, pus-containing gall-bladders to find after evacuating them that very large portions of their walls may be cut away, and even then there is plenty of tissue remaining to allow the gall-bladder stump to be embedded in the upper angle of the abdominal incision and retained there by sutures without any undue tension.

Certainly, the rapid and satisfactory recovery of the six patients on whom I performed complete cholecystectomy is a striking contrast to the troublesome biliary fistula which used to be met with in the early days of cholecystotomy; but when one learns from published statistics of the very high mortality of cholecystectomy as compared with cholecystotomy it makes me hesitate to perform it.

However, surgeons are beginning to realise that the excision of the gall-bladder will prove to be justifiable in many cases where formerly we were content with a cholecystotomy.

The Muscular Factors concerned in Ankle-Clonus.—S. Weir Mitchell recently observed in a case of trauma of the spinal cord that the very remarkable ankle-clonus present was the work of the soleus muscle alone, the gastrocnemius being entirely passive. Further observation of other cases showed that the first one was not exceptional, as in all of them the soleus alone was active in clonus of the ankle. The explanation of the lack of response on the part of the gastrocnemius appears to consist in the fact that, in spite of their common insertion, the soleus and the gastrocnemius have distinct purposes, as stated in Duchenne's 'Electro-Physiologie': "When the leg is flexed on the thigh, the gastrocnemius is so relaxed by reason of its upper hold on the femur as to lose almost all power to extend the foot. The soleus, then, because of its attachment to the tibia, is still left competent to extend the foot." Another and singular fact, which is possibly involved in the absence of clonic response by the gastrocnemius, is that there is normally a difference in irritability between different muscles. Hence, one additional reason why the gastrocnemius cannot be made to respond in any position may be that it is simply less excitable than the soleus.—*Journal of Nervous and Mental Diseases; Medical Record*, July 5th, 1901.

Syphilitic and Diphtheritic Sore Throat.—Petges concludes that pseudo-membranous syphilitic angina is not rare, that bacteriological examination alone assures a correct diagnosis, and that antitoxin should be given to all doubtful cases at once.—*Philadelphia Med. Journ.*

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THE SURGICAL TREATMENT OF FIBRO-MYOMATA OF THE UTERUS, WITH ILLUSTRATIVE CASES.*

By ARTHUR E. GILES, M.D., B.Sc.Lond., F.R.C.S.,

Gynæcologist to the Tottenham Hospital; and Surgeon to Out-patients, Chelsea Hospital for Women.

(*Concluded from p. 224.*)

(*b*) Abdominal Operations.

1. ABDOMINAL MYOMECTOMY.—By this is meant the removal, after opening the abdomen, of a pedunculated subperitoneal myoma. The uterus is left intact. The pedicle is often a narrow one, and can be dealt with in the same way as the pedicle of an ovarian tumour. In other cases the base of attachment is broader, and is then best dealt with by incising the capsule of the tumour all round, close to the pedicle, and shelling out the tumour. The pedicle is then brought together by mattress sutures, and the peritoneum united over it. This is a very satisfactory operation, as the uterine functions are in no way interfered with. It may be observed that not infrequently operation is undertaken in these cases in the belief that an ovarian tumour is present, as the usual classical symptoms of myoma are absent.

This type is shown in Fig. 4, and is illustrated by two cases.

CASE 6.—A. R—, æt. 39, single, was admitted on August 14th, 1900. She had noticed a swelling in the abdomen for twelve months, and had had pain from it. Menstruation had been regular, and she had no leucorrhœa. On admission a firm swelling could be felt in the hypogastrium, inclining towards the right side. It could easily be moved independently of the uterus. On August 15th the abdomen was opened and the tumour delivered into the wound; it was found attached to the top of the fundus of the uterus. Its base was incised near the pedicle, and the tumour enucleated; after which the pedicle was secured by mattress sutures, and the peritoneal edges brought together and sewn over with fine silk. A smaller fibroid was found growing from the right side of the fundus, and was dealt with in the same way. The uterine fundus was fastened under the anterior abdominal wall and the wound closed. Convalescence was uninterrupted, and the patient went home three weeks after operation. Microscopic examination of the tumour showed it to be an almost pure fibroma.

CASE 7.—L. I—, æt. 63, single, was admitted on February 18th, 1902. Menstruation was regular till the age of fifty-three, when the menopause occurred. Two years ago she had some hæmorrhage, which lasted two or three months; this recurred during the winter of 1900-1, and also this winter (1901-2). She had had some gnawing pain in the epigastrium. She was a patient of Dr. Marjori-

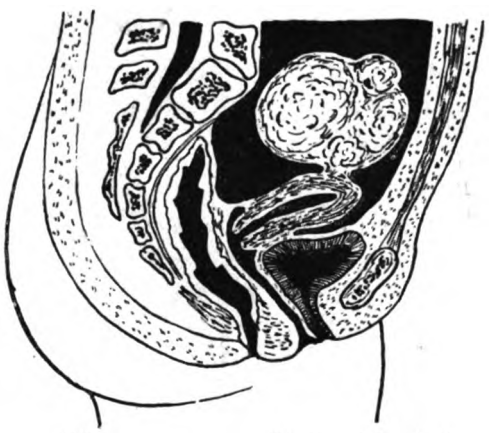

FIG. 4.—Fibro-myoma removable by abdominal myomectomy.

banks. On admission a small rounded tumour could be felt rising out of the true pelvis. The vagina was senile, conical, with the apex upwards. Cervix small and atrophied. The body of the uterus was enlarged, as if it contained an interstitial myoma; attached to it was a tumour the size of an orange, and freely movable, which appeared to be a pedunculated myoma. On opening the abdomen on February 20th the uterus was found to be enlarged, and to contain a myoma in its posterior wall. Attached to the cornu by a fleshy pedicle was a tumour the size of an orange, and partly cystic. In addition a smaller myoma was attached by a small pedicle to the anterior wall of the uterus. The pedicle of the latter was ligatured *en masse* and divided. The large and partly cystic myoma was removed like an ovarian tumour, the

* Remarks made, with demonstration of specimens, before the North-East London Clinical Society, Tottenham Hospital, on May 1st, 1902.

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pedicle being ligatured piecemeal and an encircling ligature tied round the whole; after which the stump was turned in and the peritoneum sutured over it. An incision was next made in the posterior wall of the uterus, and the contained myoma shelled out, the wall being brought together again by two mattress sutures. Convalescence was normal, and the patient went home on the nineteenth day.

2. Abdominal Enucleation.—This means the removal, by the abdominal route, of one or more interstitial myomata from the substance of the uterine wall. In some cases many separate tumours have been thus removed, sometimes through one incision. In my opinion the applicability of the operation is limited to cases where at most two or three tumours are present. In other

Fig. 5.—Fibro-myomata removable by abdominal enucleation.

cases hysterectomy is preferable, because the removal of many tumours is accompanied by a great loss of blood, and the maimed remnants of such a uterus are of no possible use to the patient. Moreover, the fact that many myomata have been present renders it probable that other tumours may form. When only one or two moderate-sized tumours have been enucleated, the uterus is still capable of the function of child-bearing. The type is shown in Fig. 5 and is illustrated by the two following cases. It is to be noted that the myomectomy was incidental in both cases, operation being undertaken on account of chronic retroversion with fixation of the uterus.

Case 8.—M. S—, æt. 36, married, was admitted on June 21st, 1901. Though married twelve years she had had no children. For six months she had had pain in the abdomen, especially on the left side, and xanthorrhœa. Micturition had been painful. Menstruation was regular but scanty. On admission the uterus was retroverted and flexed, and bound down by adhesions in the pouch of Douglas. On opening the abdomen on June 25th the appendages of both sides appeared normal. The uterus was freed of adhesions and brought forward, when it was found to contain an interstitial myoma in the anterior wall. This was incised and the growth enucleated. The uterus was then fixed to the anterior abdominal wall by three silkworm-gut sutures. Convalescence was uninterrupted, and the patient went home well on the twenty-second day.

Case 9.—A. H—, æt. 46, widow, was admitted on October 22nd, 1901. She had had no children. For two years she had had bearing-down pain, difficulty with defæcation, and painful micturition. She had been treated for seven months in the out-patient department, the uterus being retroverted and flexed, and fixed by adhesions. As palliative treatment failed to relieve her, she was admitted with a view to separation of adhesions and ventro-fixation. She was under the care of Dr. Hutt. On opening the abdomen on October 24th the fundus was found adherent in the pouch of Douglas, with a membranous band of adhesion passing from its anterior surface to the front of the sacrum. This was separated and the uterus lifted forward, when an interstitial myoma was found projecting from its posterior surface. The fundus was secured by a silkworm-gut suture and pulled well forward; the posterior wall was incised and the tumour enucleated. It was the size of a hen's egg. The wall was closed by a series of interrupted sutures, the fundus was united to the anterior abdominal wall by three silkworm-gut sutures and the wound closed. Convalescence was uninterrupted, and the patient went home on the twentieth day.

3. Abdominal Hysterotomy. — By this is meant the opening of the uterine cavity by an incision made through the abdomen, and the extraction of a myoma from its interior. It is specially indicated in the case of a single myoma occurring in a young subject. Thus I performed the operation on a single woman æt. 23 (*vide* 'Obst. Trans.,' vol. xlii, 1900, p. 299). As I pointed out in the report of that case, it is important that the cervical canal should be dilated to

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secure free drainage. Fig. 6 shows this type, of which the following example occurred recently in the present series.

CASE 10.—P. T—, æt. 31, married, was admitted on April 9th, 1902. Five years ago she had a severe nervous shock, and since then had suffered from progressively increasing menorrhagia. She had been married one year and nine months, and had had no pregnancy. For the last six months the menorrhagia had been worse, the flow lasting five days and necessitating thirty-six diapers. Before and during menstruation she was quite prostrate and unable to get about. She was under the care of Dr. David Petty. On admission the patient was exceedingly blanched and anæmic. A round firm regular tumour could be felt in the abdomen, rising out of the pelvis and reaching midway

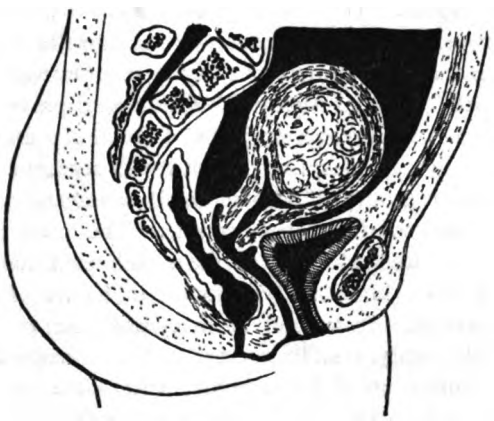

FIG. 6.—Fibro-myoma removable by abdominal hysterotomy.

between the umbilicus and the pubes. The condition was diagnosed as one of intra-uterine myoma, probably becoming polypoid. On April 10th, with the object of removing the growth *per vaginam* if possible, the cervix was dilated and a finger introduced into the uterus. A large myoma was found projecting into the uterine cavity from the posterior wall. As it was too large to remove through the cervix, the uterine canal was lightly packed with gauze and the abdomen opened. The uterus was lifted forward, a three-inch incision made through its posterior wall, and the myoma shelled out. It was the size of a closed fist. Bleeding was controlled by very hot sponges, and an aperture found between the bed of the tumour and the uterine cavity was enlarged to allow free drainage. The uterine wall was brought together as in a Cæsarean section by a series of mattress sutures, and the peritoneal edges sewn together by a continuous suture of fine silk. The uterus was slung up to the anterior abdominal wall by two silkworm-gut sutures, and the wound closed in the usual way. Convalescence: except for an attack of diarrhœa on the eighth and ninth days, the patient made a smooth recovery, and went home on the nineteenth day.

The three procedures last described are of a typically conservative character, the tumour being removed and the uterus preserved. In the remaining ones the uterus is of necessity sacrificed with its tumour or tumours.

4. ABDOMINAL HYSTERECTOMY. — By this is meant the removal of the uterus through an abdominal incision. It is generally understood that

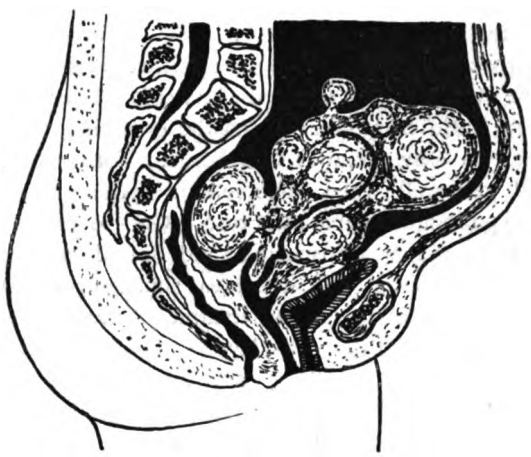

FIG. 7.—Fibro-myomata requiring abdominal hysterectomy.

the cervix is left behind, the uterus being amputated at about the level of the internal os, in contradistinction to panhysterectomy, in which the cervix also is removed. The old plan of the *serre nœud* with fixation of the stump in the abdominal wound has been abandoned by modern surgeons. Instead of this, the peritoneal flaps are sewn over the stump, and this is allowed to fall back into the pelvic cavity. All the cases that follow were operated upon on account of one or more of the indications enumerated at the beginning of the paper. It is of interest to note that two of the patients were sisters, and that I had already performed a hysterectomy for myoma on a third sister. Case 17 is of interest as being an instance of myoma affecting a double uterus. It is the only case of the kind that I have met with or seen recorded.

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The type is shown in Fig. 7, and the illustrative cases are as follows.

CASE 11.—B. N—, æt. 56, married, was admitted on April 19th, 1901. She had had four children, the youngest of whom was twenty-eight years old. She had never ceased menstruating, and for three years had had excessive loss. Two years previously a fibroid polypus was removed from the cervix, but the abatement of hæmorrhage was only temporary. She had had very little pain, and had been under the care of Dr. Gordon Roberts. On admission the uterus was hard, and enlarged to the size of a fœtal head. The patient was pale and had a systolic murmur over the pulmonary area. The abdomen was opened on April 23rd; the right appendages were ligatured off and not removed. The left were removed with the uterus, as the left ovary was cystic. The cervix was cut across low down, the uterine arteries ligatured, and the stump closed over with fine silk. There was very little hæmorrhage, and the operation, which was an easy one, was well borne. For two days all went well; then some icterus developed, and the pulse and temperature rose. On the fifth day the wound was inspected and found healthy. Some vomiting and diarrhœa supervened. She gradually sank in spite of treatment, and died on the morning of the sixth day.

CASE 12.—R. R—, æt. 43, married, was admitted on July 1st, 1901. She had had one child twenty-five years previously. For some time the catamenia, although regular, had been excessive. Two years ago she was in the Soho Hospital, where a polypus was removed; the loss nevertheless continued, the periods lasting ten or twelve days. She had had abdominal pain, but no interference with bladder or bowels. She had been under the care of Dr. Murison, of Erith. On admission the patient looked pale and anæmic. On vaginal examination the uterus was enlarged to the size of a three months' pregnancy, and a hard mass was found to the left, apparently in the broad ligament. On opening the abdomen on July 2nd both ovaries were found cystic, and these were removed first. A large mass had burrowed into the left broad ligament, and this was carefully enucleated, after which the uterine arteries were tied, and the uterus removed with its contained myoma by cutting across the cervix. Oozing was rather free. The stump was drawn together by mattress sutures, and the peritoneum sewn over it. A hot enema of normal saline solution to one pint was administered on the patient's return to bed. Convalescence was quite satisfactory, and the patient returned home on the twenty-fifth day.

CASE 13.—L. C—, æt. 40, single, was admitted on July 26th, 1901. For five years she had been troubled with pain in the lower part of the abdomen, and difficulty in passing water. The latter symptom had lately increased in severity, and on several occasions just before admission she had had retention of urine, necessitating the use of a catheter, on account of which she sought the aid of Dr. Carter, of St. John's, S.E. On admission a large hard tumour occupied the lower part of the abdomen. Vaginal examination showed that the tumour completely filled the pelvis, lying behind the vagina. The finger could hardly be passed between the tumour and the pubes, and the cervix was drawn up almost out of reach. On opening the abdomen on August 6th the tumour proved to be one large myoma springing from the posterior part of the fundus, from which it was separated by a slight groove. Several small myomata were attached to the uterine cornua. The mass had become wedged down in the pouch of Douglas, and when lifted up was found to be free from adhesions. It formed an accurate cast of the pelvic cavity, and its surface was well marked by an impression of the sigmoid flexure. The uterus was amputated just above the internal os; there was hardly any bleeding, and the operation was an easy one. The appendages were healthy and were left behind. Convalescence was interrupted only by a little rise of temperature from the third to the seventh day, associated with some effusion in the right broad ligament. This soon subsided, and the patient went home well on the twenty-fourth day.

CASE 14.—E. H—, aged 33, married, was admitted on November 13th, 1901. She had had no children and had been married four years. Ten days before admission she was seized with violent pain in the abdomen, and her doctor advised her removal to hospital. On admission a very hard nodular tumour was found rising from the pelvis; it was the size of a melon, was irregular in outline, and had small nodular excrescences over its surface. It reached to the umbilicus and inclined to the right. The cervix was normal and

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nulliparous. The uterus was lying apparently retroverted and partially fixed. In front was the large hard tumour lying obliquely, the upper part being to the right and the lower part to the left. On opening the abdomen on October 24th the tumour was found to be a large myoma attached to the anterior surface of the fundus. When it was cut away the uterus was found to contain a number of interstitial myomata. This and the presence of hydroperitoneum led to a decision to remove the uterus. This was rendered very difficult, as the anterior surface of the uterus was firmly attached to the bladder as high up as the fundus by very vascular adhesions. These were separated, and the bleeding with difficulty arrested. The left ovary was cystic and was removed, and the uterus cut away after ligature of the uterine arteries. Much loss of blood was caused by numerous punctiform hæmorrhages, and the operation lasted an hour and a quarter. Transfusion of saline solution was done on the table. The patient rallied well, and recovery was uneventful, so that she went home well on the twentieth day.

Case 15.—E. C—, æt. 45, single, was admitted on November 26th, 1901. I had previously performed abdominal hysterectomy for myoma on two of her sisters, one of whom was Case 13. Two or three years before admission she noticed a hard movable swelling in the left iliac region. Since then she had had sharp cutting pains during her periods. Micturition had been difficult for the last year, and especially so for the last four or five months. On lying down she had constant desire for micturition, with pain after the act. She had to rise three or four times during the night. On admission a large, firm, pear-shaped tumour was found, rising out of the pelvis and reaching to within an inch of the umbilicus. The vagina was long and narrow; the pelvis was filled up with a firm rounded tumour, which was slightly movable. The cervix was high up and far back. On opening the abdomen, on November 28th, a large myomatous uterus presented. It was bound down on the right side by the appendages, which were adherent to the broad ligament. The arteries on the left side were secured, the tube ligatured and divided. The right side was with great difficulty treated in the same way. The peritoneum was reflected down in front and behind, and the cervix cut through. The right uterine artery was difficult to ligate on account of the condition of the right appendages. The uterus being removed, the right ovary was found to be cystic, the size of a turkey's egg. In breaking down its adhesions it burst, and flooded the pelvis with thick black fluid. It was removed, and proved to be a dermoid cyst. The operation was completed in the usual way. Convalescence was normal, and the patient went home on the twenty-third day.

Case 16.—A. B—, æt. 46, married, the mother of a daughter twenty-one years old, was admitted into hospital on February 10th, 1902. Menstruation had always been regular, and attended with dysmenorrhœa and menorrhagia. For twelve months, however, she had had irregular menstruation; and profuse losses attended with the passage of clots had occurred within the last three months. She was under the care of Dr. MacVine. On admission there was a firm, regular, rounded swelling filling the pelvis and rising into the abdomen, to midway between the umbilicus and the pubes. The vagina and cervix showed senile changes. On opening the abdomen, on February 13th, the tumour at once presented. The right ovary was large and cystic; it was therefore decided to remove it; a ligature was placed on its distal aspect, and the broad ligament cut down till the uterine artery was seen. On this a forceps was placed. The left side was dealt with in the same way, except that this ovary was healthy and was left behind. The cervix was then cut across. There was some hæmorrhage from a branch of the uterine artery which had divided low down; but this was secured and a satisfactory bloodless stump was obtained. The peritoneal margins of the broad ligament were sewn together from end to end over the stump, which had been braced together with two mattress sutures. The wound was united in three layers. Convalescence was normal, and the patient went home on the nineteenth day.

Case 17.—E. E—, æt. 53, single, was admitted on December 15th, 1901. Menstruation started at the age of eighteen, and had continued regularly till the present time. Four months ago she began to have some pain in the right side of the abdomen. It was dull, and ran down the right leg. She had had great difficulty with defæcation and was very constipated. Pain had gradually increased, and was only relieved by the recumbent position. On admission the abdomen was pro-

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minent and irregular, bulging in the right iliac and umbilical regions. On palpation there was a large tumour occupying the whole of the pelvis, and rising into the abdomen to the level of the umbilicus. *Per vaginam* the hymen was unruptured, thickened, and fibrous, and the orifice so small that no proper examination could be made. On opening the abdomen, on December 17th, the tumour proved to be a large myomatous uterus. The left ovary was normal and was left behind. The ovarian arteries and the round ligaments having been secured and ligatured, the broad ligament was divided on either side. The peritoneum was reflected down in front and behind, the uterine arteries ligatured, and the cervix cut across. A mattress suture was put in the stump, and the peritoneum sewn over it from side to side. The right ovary being cystic was removed with the uterus. The tumour weighed four and a half pounds, and proved to be a myomatous double uterus, of the form known as uterus septus. Convalescence was uninterrupted, and the patient went home on the twenty-third day.

5. Abdominal Panhysterectomy.—By this is meant the complete removal of the uterus, including the cervix, through an abdominal incision. There are several ways of performing the operation. The plan I have followed is that of Mr. Bland-Sutton, who, either after cutting across the uterus, or omitting that step, dissects the cervix out, leaving only its vaginal covering, which thus resembles a tailor's thimble from which the finger has been removed. The advantage of this procedure is that there is less likelihood of effusion in the neighbourhood of the stump; but there may be a little difficulty caused by bleeding from the inside of the thimble-like cap of cervix. This, however, can generally be controlled. Whether this or the previous method be adopted depends largely on the suitability of the individual case.

Panhysterectomy was performed in the two following instances.

Case 18.—M. S—, æt. 48, married, was admitted on January 7th, 1901. She had not been pregnant, though married seven years. Two years before admission she began to suffer from excessive hæmorrhage at the monthly periods. In July, 1900, I saw her in the out-patient department, and ordered styptics. Improvement was only temporary. On one occasion she had difficulty of micturition at the monthly period. She had been under the care of Dr. Marjoribanks. On admission a large tumour was found rising out of the pelvis into the abdomen and markedly lobulated. The cervix was lying behind the pubes, being pushed forwards by a mass behind and to the right of the uterus. On opening the abdomen on January 15th a mass of multiple fibroids was found connected with the uterus. Both ovaries were diseased, and were removed together with the uterus and the whole of the cervix, a panhysterectomy being performed. The operation, which lasted forty-five minutes, presented no difficulties. Convalescence was quite satisfactory, and the patient left for the seaside on the twenty-seventh day.

Case 19.—M. A. D—, æt. 51, single, was admitted on February 14th, 1901. For seven or eight years she had had excessive losses of blood, from which she felt faint and weak. On admission the patient was exceedingly pale and sallow with anæmia. On examination the pelvis was filled up with a large hard mass connected with the uterus. She was kept in bed for three weeks, and treated with iron, ergot, and cotarnine, to endeavour to improve her general condition. The loss, however, continued freely, and it was felt that her only chance lay in surgical intervention. On opening the abdomen on March 5th a large subperitoneal fibroid was found attached to the right side of the fundus; the uterus itself was very large. The ovary on the right side was diseased, and was removed. Preventive ligature of vessels was adopted, and the cervix was cut out as described. The loss of blood was very slight, hardly more than a drachm altogether, and the operation was well borne. Convalescence was slow. A suture abscess formed after a few days, and continued to discharge for about three weeks. This eventually healed, and she went home in good condition seven weeks after operation.

6. Cæsarean Hysterectomy.—I have put this in a category by itself, because the indications for operation are not those that usually apply to myomata, but depend rather on obstetric considerations. The operation is undertaken because labour is obstructed by one or more myomatous tumours. The steps of the operation resemble those of an ordinary hysterectomy, except that the child and placenta must first be removed by Cæsarean section. Hence I have called it

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Cæsarean hysterectomy. It differs from Cæsarean section proper in that the uterus itself is sewn up and left behind in the latter operation; and it differs from Porro's operation because the features of the latter are the use of the *serre nœud* and the extra-peritoneal fixation of the stump in the abdominal wound. The type is shown in Fig. 8, and I am able to describe an illustrative case which occurred quite recently in the hospital.

Case 20.—T. L. H—, æt. 31, was admitted on April 17th, 1902. She had been married eighteen months, and was in labour of her first child at full term. Labour started at 4 a.m. on the day of admission. At 2 p.m. the membranes ruptured.

Fig. 8.—Fibro-myoma obstructing labour, removed by Cæsarean hysterectomy.

At 6 p.m. her medical attendant, Dr. Hutt, was called in. He found the os almost out of reach behind the symphysis, and the cord prolapsed. There was an indefinite tumour in the posterior wall of the lower segment, which was blocking the way of the head. A shoulder was presenting. He sent for Dr. Chappel, who administered chloroform; the head was pushed up, and after some difficulty a leg was brought down, but all advance was blocked by the tumour behind. The pains were feeble and infrequent. Dr. Hutt advised that she should be at once removed into the hospital, and I was immediately summoned. On admission the patient was very nervous and excited. The pulse rate was 100. On my arrival at 10 o'clock there was a leg in the vagina, the foot projecting outside the vulva and pointing backwards. The os was high up and jammed against the pubes. Behind the cervix was a large hard tumour, which could not be moved, and so blocked up the inlet of the pelvis as to allow an interval of only one and a half inches between it and the pubes, this interval being filled up by the leg.

A diagnosis of subperitoneal myoma or solid ovarian tumour was made; the child had been dead about two hours, but it was deemed impossible to remove it *per vias naturales*, even by embryotomy, and abdominal section was decided upon. The operation was undertaken at once. A seven-inch incision was made from three inches above to four inches below the umbilicus, and the uterus was at once brought outside the wound. The hand was passed down behind, and the tumour was found to be a large myoma springing from the posterior wall of the uterus low down. It was impacted under the sacral promontory, and adherent to the floor of the pelvis. As, in addition, some myomata were found studded over the anterior surface of the uterus, it was at once decided to extract the child and do a hysterectomy. Accordingly the uterus was incised in the middle

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line, the child delivered, and the placenta and membranes shelled out. The uterus was compressed with the hands and a swab placed in its cavity. The broad ligaments were tied off and divided, the uterine arteries seized with forceps, the peritoneum and bladder reflected down from the front of the uterus, and the cervix cut across. The uterus was then attached only by the large myoma; a circular incision was made through the capsule of the latter and the tumour shelled out. The arteries were then tied, and the walls of the widely dilated cervix approximated by two mattress sutures. The adhesions fastening down the capsule of the myoma were then broken down and the capsule removed. A hot sponge was efficient in checking the bleeding. The peritoneal layers of the broad ligament were sewn together over the stump. No trace of the right ovary could be seen. The left was healthy and was left behind. A pint of normal saline solution was poured into the peritoneal cavity, and the wound closed in three layers. During the operation the patient was at one time much collapsed; she was given $\frac{1}{15}$ gr. of strychnine and 15 min. of tinct. digitalis subcutaneously. She was also given a subcutaneous injection of two pints of normal saline solution under the breasts. On her return to bed she had a brandy-and-beef-tea enema, and this was repeated four-hourly. Two hours after the operation the patient had rallied very well. Convalescence was very satisfactory; the temperature remained normal throughout, and there were no complications. The patient got up on the fourteenth, and went home on the twentieth day after operation.

The object of this paper has been to indicate the widely different methods that are applicable to the surgical treatment of myomata, and to emphasise the fact that no single plan is by any means appropriate to the treatment of all cases, each of which requires to be dealt with individually on its merits. Of course, the key-note of the whole thing is conservatism, by which I understand the preservation of organs, in so far as this is compatible with the immediate safety and the subsequent health of the patient. Only one ultimate aim is permissible, namely, the cure of our patients; and by this result our work must stand or fall.

The summaries of the cases recorded are based on the excellent notes of our successive resident medical officers, Mr. Lionel Provis, Dr. G. Norman Meachen, and Dr. C. Lawson Smith.

Tabular Statement of Cases operated upon for Myoma in the Tottenham Hospital, March, 1900—April, 1902.

No.	Name.	Age.	Married, single, or widow.	Medical attendant.	Date.	Diagnosis.	Operation.	Result.
1	E. M.	42	M.	—	Oct. 25, 1901	Intra-vaginal myoma	Vaginal myomectomy	Recovered.
2	J. J.	47	M.	—	April 12, 1902	,,	,,	,,
3	J. P.	38	M.	—	June 9, 1900	Intra-uterine myomata	Vaginal enucleation	,,
4	F. T.	43	M.	—	March 18, 1900	Large intra-vaginal myoma	Vaginal myomectomy with morcellement	,,
5	E. M.	38	M.	—	May 22, 1901	,,	,,	,,
6	A. R.	39	S.	—	Aug. 14, 1900	Large subperitoneal myoma	Abdominal myomectomy	,,
7	L. I.	63	S.	Dr. Marjoribanks	Feb. 18, 1902	Multiple subperitoneal myomata	,,	,,
8	M. S.	36	M.	—	June 21, 1901	Interstitial myoma	Abdominal enucleation	,,
9	A. H.	46	W.	Dr. Hutt	Oct. 22, 1901	,,	,,	,,
10	P. T.	31	M.	Dr. Petty	April 9, 1902	Large intra-uterine myoma	Abdominal hysterotomy	,,
11	B. N.	56	M.	Dr. Gordon Roberts	April 19, 1901	Large uterine myoma	Abdominal hysterectomy	Died.
12	R. R.	43	M.	Dr. Murison	July 1, 1901	,,	,,	Recovered.
13	L. C.	40	S.	Dr. Carter	July 26, 1901	,,	,,	,,
14	E. H.	33	M.	—	Nov. 13, 1901	,,	,,	,,
15	E. C.	45	S.	—	Nov. 26, 1901	,,	,,	,,
16	A. B.	46	M.	Dr. MacVine	Feb. 10, 1902	,,	,,	,,
17	E. E.	53	S.	—	Dec. 15, 1901	Large myoma in a uterus septus	,,	,,
18	M. S.	48	M.	Dr. Marjoribanks	Jan. 7, 1901	Large uterine myoma	Panhysterectomy	,,
19	M. D.	51	S.	—	Feb. 14, 1901	,,	,,	,,
20	T. H.	31	M.	Dr. Hutt	April 17, 1902	Large uterine myoma obstructing labour	Cæsarean hysterectomy	,,

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THREE CLINICAL LECTURES ON THE DIAGNOSIS AND TREATMENT OF RECTAL DISEASES.

Delivered at the Medical Graduates' College and Polyclinic, May 26th, June 2nd and 9th, 1902.

By F. C. WALLIS, F.R.C.S.,

Assistant Surgeon to Charing Cross Hospital and to St. Mark's Hospital.

LECTURE I.

IT is common knowledge that few people go through life without some rectal trouble. It is therefore essential that the main points in the ordinary diseases of the rectum should be well known to all of us, but it is quite unusual to find this the case, and ignorance is the rule rather than the exception in the matter of rectal surgery. The reason for this undoubted fact is that when working in hospitals, either as dressers or house surgeons, the natural tendency is to take interest in grave cases demanding big operations, and thus such cases as those which I propose now to discuss are either entirely ignored or treated in a most perfunctory manner. The consequence is that when the time for actual practice comes most medical men and a large number of surgeons find themselves quite ignorant of all or most rectal ailments, which they also find comprise a large percentage of their ordinary practice.

Having had now for some years special opportunity at St. Mark's for studying this branch of surgery, I thought I could not do better than take this subject for the three lectures your secretary has courteously asked me to give. The order in which I propose to take the various diseases in the first lecture differs from the stereotyped form because I believe, without wishing to anticipate too much, that many of the common rectal troubles have a common origin, namely a simple ulcer, to which I shall have to refer a great many times.

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Before going into symptoms and diseases it is necessary to consider superficially the development of this portion of the anatomy.

The method of fusion of the proctodeum and the blind end of the gut has markedly to do with the circulation in this area, and also with the low vitality of some of it, and with the folds of mucous membrane which exist, and to which various names are given. The frequency and common situation of the ulcer which I have mentioned have also to do with this. The distribution of the three hæmorrhoidal arteries is briefly as follows:—The inferior hæmorrhoidal, a branch of the internal pudic, supplies the skin and muco-cutaneous junction. The middle hæmorrhoidal, coming from the internal iliac, supplies the muscles and subcutaneous tissue around the bowel. The superior hæmorrhoidal is a branch of the inferior mesenteric, and supplies the original intestine, which ends at about the situation of the internal sphincter. It is here that the hæmorrhoidal plexus—a frequent source of trouble—exists. Immediately below this, that is the aforesaid plexus, the lining membrane of the rectum differs somewhat in character from ordinary mucous membrane, and at this point, namely between the two sphincters, it will be found that nearly all the ordinary troubles known as rectal diseases originate or exist; and this undoubted fact is, I believe, due mainly to the circumstance that the blood-supply in this area is comparatively small, and, in spite of a free collateral circulation existing in the surrounding tissue, the absolute lining membrane of the junction of the proctodeum with the bowel is quite scantily supplied with blood.

The nerve-supply of the anus and the rectum is derived from the branches of the sacral plexus, of which the inferior hæmorrhoidal and the superficial perineal nerves form an important part. The final curve of the rectum should be borne in mind; also the fact that the fusion spoken of takes place generally between the sphincters.

Pruritus Ani.

The symptoms of pruritus ani are probably well known to most of us, but it is worth while to enumerate them before going on to the causes and treatment. The main symptom is intense itching, which becomes accentuated at night-time when the patient is falling asleep, and the persistence of this most distressing symptom causes a very marked effect upon the life of the individual sufferer. It is obvious that the constant irritation occurring night after night, preventing sleep, very soon brings in its trail other symptoms and troubles due to want of sleep. Inability to work, irritability of temper, dyspepsia, and loss of appetite are the concomitants of this trouble.

The local appearance of the area affected in pruritus ani is quite typical, and should be at once recognised as pathognomonic of the trouble. There are two varieties, the dry and the moist. The reasons for the one or the other are probably largely a matter of some other idiosyncrasy of the patient, such as gout and the like. The *external* treatment varies somewhat according to which variety exists. On inspection and comparing the affected area with the normal condition it will be found that the skin margin in the neighbourhood of the external sphincter has completely altered in character. I am taking a chronic case because it shows the main symptoms. Chronic irritation and constant scratching produce a chronic inflammation of the skin, so much so that, as in other parts of the body, the eventual result is a complete alteration in the character of the skin, which in itself ceases to be true skin and becomes epidermis, covering the remains of true skin largely infiltrated with fibrous tissue.

A number of causes have been assigned to pruritus ani. And various treatments, even more assorted than the assigned causes, have been adopted, but the results of the ordinary treatments of pruritus ani are in the main most disappointing except in slight cases, which are due usually to some error in diet. The trouble in itself is chronic; the usual treatment, even when in rare cases it eventually effects a cure, is so prolonged that often before this desired point is arrived at the patient gets tired of it and loses confidence in his adviser, and either lapses into his former condition or seeks advice elsewhere.

The chronicity and great frequency of these cases in the out-patient department at St. Mark's Hospital made me investigate for some possible cause. It was no uncommon thing to have between fifteen and thirty patients in an afternoon, some old and some new, suffering from pruritus ani. The old cases came regularly month after month, and in some cases year after year. They were given

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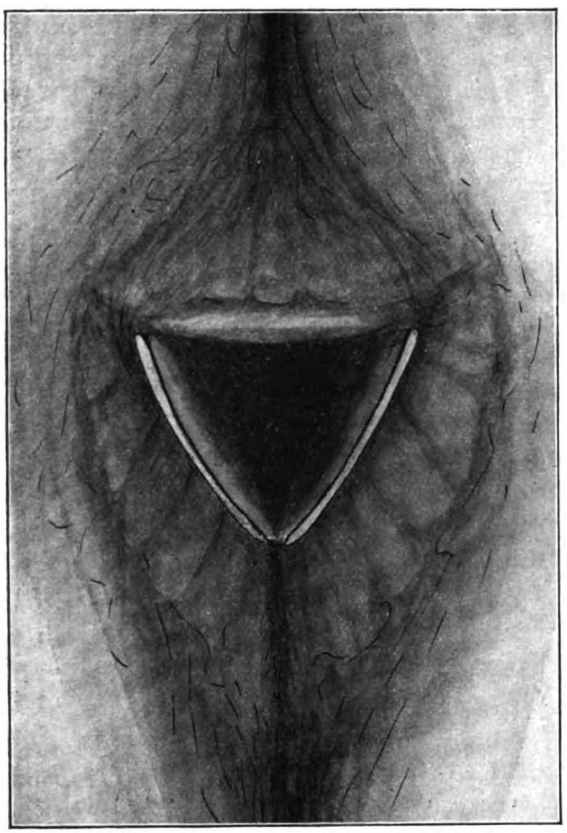
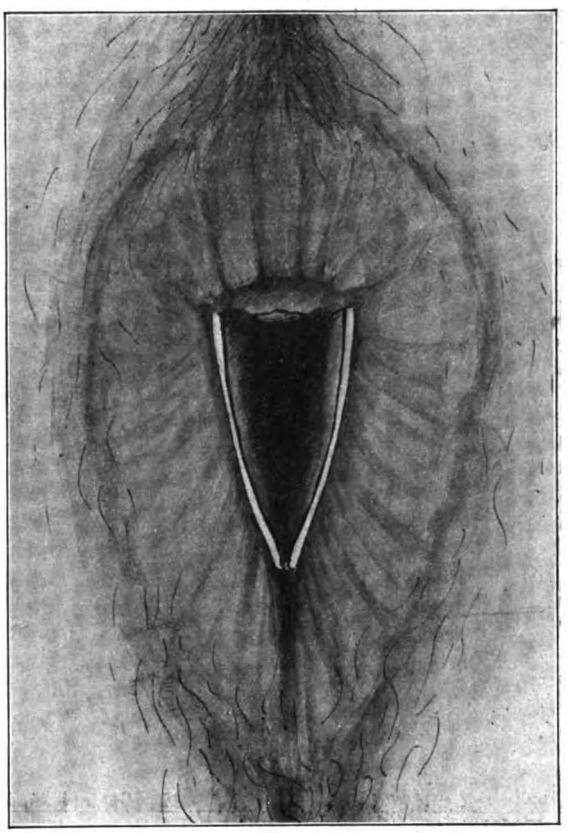
a variety of ointments and medicine, but I cannot say that any but the very smallest amount of success followed the treatment. It was under such circumstances that I started investigating for a cause, and about three years ago I made a point of examining every case of pruritus ani, both digitally and by means of a speculum. In a large number of cases, that is well over 50 per cent., I found a shallow ulcer situated between the two sphincters, and more often in the posterior half

FIG. 1.



than in any other part of the circumference. This ulcer is not easy to recognise by the touch, and it requires a certain amount of practice to appreciate its presence. In the first place it must be remembered that it is only just within the anal margin, and always below the internal sphincter. The smooth feeling of the healthy mucous membrane will be recognised, but when the finger comes on to this abraded or ulcerated surface the smooth feeling disappears, a slightly raised margin can be felt, and the area beneath the finger inside this raised margin is felt to dip under the touch. There is sometimes pain, but more often none, associated with the examination. When the speculum is introduced it must be remembered that the tissues are pushed in some little way with the introduction of this instrument, and therefore the ulcer will appear higher up than it really is. If after the introduction of the speculum it is opened to its fullest extent, the inexperienced observer will probably not recognise the ulcer (*vide* Fig. 1), but if the

FIG. 2.



speculum is only slightly opened, and a careful view with, when possible, a head-light is made, the ulcer can be clearly seen as a shallow, oval, livid abrasion (*vide* Fig. 2) differing markedly and mainly in colour from the normal pink of the mucous membrane. Having regard to the constancy of this ulcer in all these cases of pruritus ani, and failing to find any other cause of sufficient merit, I determined to treat the ulcer, believing as I did that this was the cause of the pruritus. It may be asked, why should a simple

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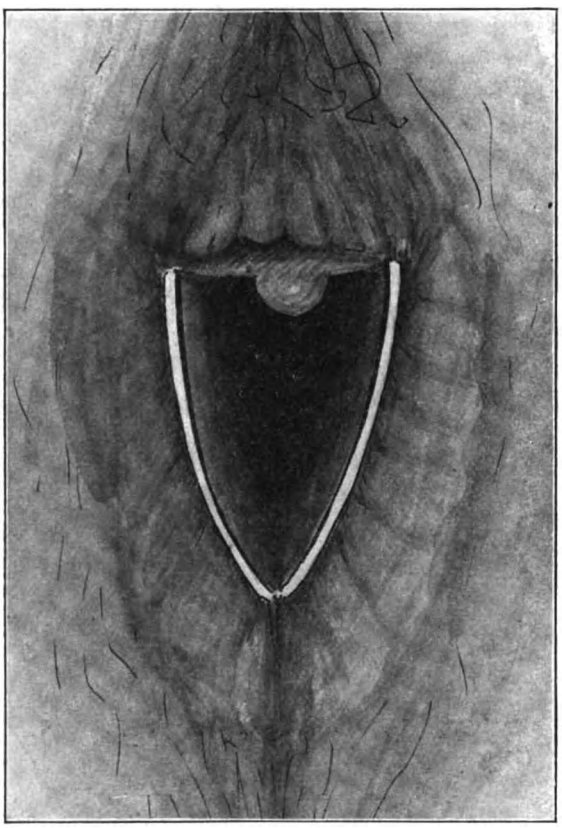
ulcer of the description given cause such symptoms? My belief is that it is due to the irritating effect of whatever secretion comes from the ulcer.

Not to weary you with the various attempts in the way of lotions, ointments, etc., which were tried, I will come at once to the most recent treatment which I have adopted with, I think, quite fair success in these troublesome cases. The ulcer is exposed by means of the speculum. A 4

FIG. 3.



per cent. solution of eucaine is then injected just behind the ulcer. This renders the area anæsthetic, and by distending the submucous tissue makes the ulcer prominent (*vide* Fig. 3). The whole of the ulcerated area is now cauterised with an electric thermocautery. The speculum is withdrawn and a morphia suppository introduced, and vaseline is applied over the cauterised area. The patient is given an astringent medicine, composed of catechu and opium, for two days. The bowels are then freely opened, and a lotion of chinosol of the strength of 1 in 500 is injected to the amount of two ounces. This injection is repeated every night and morning, and after the bowels have acted. The outside condition is best treated by an application of resorcin ointment, ten grains to the ounce, in the dry variety, and with equal parts of zinc oxide and boric acid powders in the moist kind.

Additional relief is obtained by the patient at night-time sitting in a warm bath in which is a gauze bag filled with bran, and during the night further help is obtained by keeping the apposed skin surfaces apart with some cotton wool kept in place by a T bandage. The immediate result is one of relief to the patient, but it is not unusual for some recurrence of the original symptoms to occur, and it may be necessary to have resource again to the cautery. My statistics, however, show that in the main these methods produce better results than any that have yet been tried.

Mr. Bowen, my late house surgeon, has kindly collected ninety-seven cases of pruritus ani which came under treatment whilst he was in office.

The analysis of these cases is as follows:

Number of cases	97
Number with ulcer recorded as present	53
,, ,, no record of presence or absence	35
,, ,, with fissures	4
,, ,, fistulæ	3
,, ,, worms	1
,, ,, record of definite *absence* of ulcer	1
	97

Where the ulcer has been diagnosed and treated with the cautery the improvement has been most marked and rapid. This is especially borne out in certain chronic cases, of which the two following are examples:—*Case T 200:* Treated with medicines and ointments for eleven months, when the ulcer was recognised and treated. Well in one month. *Case X 84:* Under treatment for seven months. Admitted and skin cauterised—improvement; but pruritus returned after six months, when the ulcer was diagnosed and cauterised. Attended only four times after this, and was cured.

This treatment, it must be borne in mind, has usually been carried out on hospital out-patients. who are unable to lie up.

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Fissure.

The next common complaint which I desire to discuss with you is that of fissure. It is at times one of the most painful and distressing diseases which I have met with in surgery, and the pain suffered by the patient is out of all proportion to the very slight lesion which brings about this condition. It is the one malady for which patients come asking that an operation may be done—the usual request, as we know, being that an operation shall be avoided by any means possible.

It seems in order to take this condition of fissure immediately after that of pruritus ani, because the cause is the same, namely, an ulcer, and the difference between the absence of pain in the one and the intense suffering in the other is merely a question of the situation of the ulcer. To repeat what I said with regard to the situation of the ulcer in pruritus it is between the two sphincters and well within the external sphincter. Now the ulcer in a case of fissure is situated over the external sphincter, consequently the various alterations in the contractions and relaxations of the muscle have the effect of alternately stretching and relaxing the ulcer which is situated over it. The nerve-supply to this area is quite large, and the ultimate nerve-communications are many in number.

The cause of the ulcer is, I have no doubt, the same as that of the preceding malady, pruritus, and it is merely a question of situation as to the symptoms to which it gives rise. The appearance is somewhat typical. On inspection it will be found that there is a small polypoid excrescence situated in the mid-line dorsally, usually just external to the anal orifice. If the parts are separated immediately above this small excrescence an ulcer will be seen. If an examination is now made, which, by the way, is often an extremely painful process and not immediately necessary, it will be found that this ulcer extends a small way within the bowel, and at the upper end another small polypoid growth limits its extent in this direction.

I have given you the typical appearance which is mostly described; but there are a large number of fissures which do not possess this condition of polypoid excrescence, or Vanburen's pile, as it is called.

The other marked feature in this condition is the hypertrophy of the external sphincter. This hypertrophy is brought about by the constant condition of spasm in which the muscle exists. The reason for this brings me to the symptoms. These are pain of more or less intensity at the time when the bowels are acting; but the main distress is usually afterwards, when, at varying times, from a few minutes to half an hour, and from that to an hour, the pain begins, is often of an agonising nature, and goes on for a varying time, sometimes indeed for many hours, before any relief is obtained. It is almost unnecessary to dilate upon the forlorn condition of the sufferer under these circumstances, but it must be obvious that the last thing the patient desires under such conditions is that the ordinary action of the bowels shall take place. This, in itself, brings about a very unsatisfactory condition of the large intestine, and eventually of the digestive track as a whole.

With regard to other complications which may ensue, the two worst cases of ischio-rectal abscess and subsequent complicated fistula which I have seen have started from fissures. These two cases were described by me in February, 1898, in the 'St. Bartholomew's Hospital Journal,' in a paper written on this same subject. They were, briefly, as follows:

A girl æt. 18 attended on September 16th, 1896, at the out-patients' at St. Mark's for pruritus ani and a fissure. She was given medicine, etc., and her name was put down to come in as soon as possible. She attended once after that, and then the mother came saying the girl was too ill to move. The patient was brought to the hospital in a cab, and it was at once evident that she was extremely ill. She had a temperature of 103°, a quick pulse, furred tongue, and an anxious expression. On examination, a large bilateral ischio-rectal abscess was found, and pus of the most foul odour was escaping from the bowel. The abscess was opened and the tension relieved. Two days later, under an anæsthetic, a large internal opening was discovered well above the internal sphincter. The patient was eventually discharged on April 3rd, 1897, the whole treatment extending over a period of six months.

The other case was that of a man æt. 49, who attended for a fissure three weeks previous to his admission on February 20th, 1897, when he was admitted for an almost exactly similar condition to that of the last patient. This patient was dis-

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charged on June 5th, 1897, having been in the hospital four months and a half.

I have quoted these cases because they illustrate strongly that the less delay there is in the treatment the better. It must not be imagined that every case of fissure may ultimately become complicated in this manner. But with these examples in one's mind, and having regard to the usual symptoms, which are in themselves quite bad enough, when once the trouble is diagnosed, there should be no delay in the simple and effective operation which, when properly done, invariably cures the patient. A great many varieties of somewhat fanciful treatment have been advocated from time to time, and I should like here to refer to a paragraph in the epitome of current medical literature in the 'British Medical Journal' of March 15th of this year. Here, under the heading of "The Treatment of Anal Fissure," Gussenbauer is said to advocate stretching the sphincter as being superior to all other methods. Incision is said to be a serious operation in which considerable hæmorrhage is unavoidable, and that there is danger of septic infection. Then, after describing the methods of stretching the sphincter, this final statement occurs: "Local anæsthesia of the mucous membrane is useless." If I refer to my own statistics and to those of some of my colleagues at St. Mark's, I should say as definitely as Gussenbauer almost to the exact contrary. On the few occasions that the sphincter has been dilated the result has been either a very small benefit or none at all, whereas when the sphincter has been divided, whether with or without previous stretching, the cure is absolute and practically immediate. As to the hæmorrhage being considerable, I have never seen it. Almost invariably a small branch of the inferior hæmorrhoidal is divided, but what hæmorrhage there is is easily controlled by pressure, and failing this, the application of a small quantity of adrenal extract acts immediately in checking it. As to the statement that local anæsthesia is useless, I have now for over three years used eucaine locally in hundreds of cases, and have always found that when properly used the effect is perfect. Perhaps without further controversy it would be more to the point if I briefly describe the ordinary method which I have now adopted for many scores of cases of fissure, and with the best results.

The patient is preferably placed in the genu-pectoral position. If this is not desirable the operation can be done with the patient lying on the side. The local area of skin is washed over with a strong solution of izal. A hypodermic syringe filled with 4 per cent. eucaine solution is injected one inch behind the anal margin, immediately posterior to the line of the fissure. The first injection is given subcutaneously, the next is injected somewhat nearer the fissure, and the needle is thrust deeper into the tissue and directed inwards towards the bowel. As soon as this has been completed an incision is made, commencing at the lower limit of the fissure and extending backwards along the line of anæsthesia to the full extent of the sphincter. Also, the incision is deepened until the whole thickness of the sphincter is divided. It is not necessary that the entire sphincter should be divided; the inner portion may be left intact. The incision thus made is kept apart by a plug of either gauze or cotton wool, which prevents the apposition of the cut surfaces and checks the hæmorrhage. A suppository of morphia is introduced, and a sufficient pad of wool kept in place by a firmly tied T bandage completes the operation. The patient is instructed to go home and keep quiet for two days, and during that period a few doses of the following mixture are taken:

℞ Liquoris ammonii acetatis . .	ʒj
Tincturæ catechu	ʒj
Tincturæ opii	♏xj
Tincturæ cardamomi compositi .	ʒj
Aq. cinnamomi ad	℥j

On the evening of the fourth day a somewhat strong purgative is administered, and after this has acted, the patient washes the wound area with some antiseptic solution, and another small plug is introduced into the wound, which from now on is dressed daily and kept scrupulously clean until the healing has been effected. Beyond a certain amount of pain experienced when first the bowels act after the operation, the sequence is a comparatively happy one to the patient if the bowels are kept sufficiently relaxed. The pain ceases both at the time and afterwards, and the patients, in the course of ten days to a fortnight, are healed of their wounds, and it is unusual for them to experience anything except quite transient pain after the operation. This is my experience in the out-

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patients at St. Mark's. I have seen only one out of all my cases which became somewhat septic, the rest have healed well and remained cured. The benefit which I claim for this treatment is that, given any one with ordinary intelligence and ideas of cleanliness, this operation need not keep the wage-earning man or woman or the much-occupied business man from his work for more than four or five days, and not necessarily as long as that. As to the effects of the local anæsthesia, any one who has once seen an operation done under eucaine will have no doubt of the efficacy of this most valuable drug.

Fistula (Non-tubercular) and Ischio-rectal Abscess.

Fistulæ which are not tubercular in origin are always preceded by an abscess, and a complete ischio-rectal abscess always ends in a complete fistula. At least this is my contention, and my reasons for saying so are that I have never seen an ischio-rectal abscess, properly so called which had its origin in any external source. This has been borne out after careful observation of some years, and the accompanying record of 246 cases of fistula goes to prove it.

Analysis of 246 cases of fistula in ano:

Records of previously existing ischio-rectal abscess	153 = 67 per cent.	
No record of such abscess	93	
	246	

Straight Fistulæ.

Internal opening close to or in dorsal mid-line	54	189
" " situated laterally	115	
" " close to or in anterior mid-line	20	

Horseshoe or Semi-horseshoe Fistulæ.

Internal opening close to or in dorsal mid-line	49	57
" " close to anterior mid-line	5	
No record	3	
	246	

It will be remembered that there are certain follicular troubles in connection with this area, as there are in the region of the axilla, where, as well as in the region under discussion, extensive intercutaneous suppuration will occur from septic inflammation in and around hair follicles, but this suppuration does not extend to any depth, and has none of the special features of a deep-seated ischio-rectal abscess. I must again call your attention to the ulcer, which I have already mentioned more than once. If it will be borne in mind that the situation of this ulcer is between the two sphincters, and also that its common situation is in the dorsal segment, it will be seen that the coincidence with regard to the internal openings of fistulæ is, to say the least of it, rather remarkable. With practically very few exceptions the internal opening of a fistula is situated between the two sphincters. This is a matter of common knowledge, and it is only reasonable to suppose

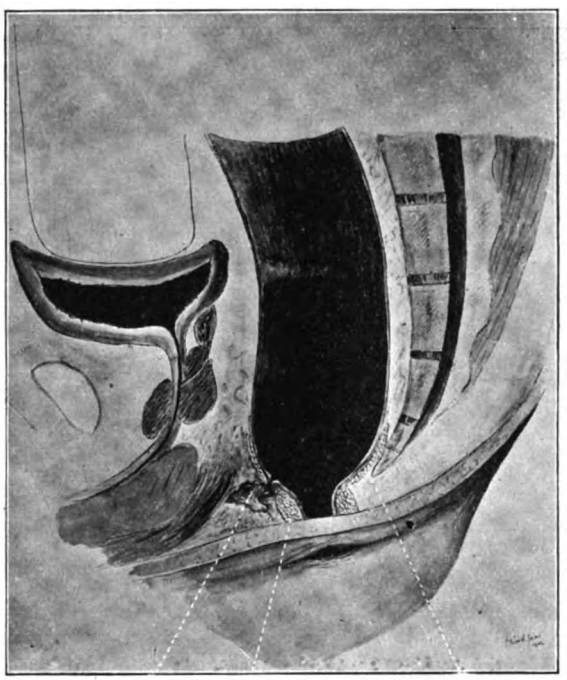

Fig. 4.—A. External sphincter. B. Internal sphincter. C. Fistula commencing from ulcers situated between the sphincters.

that given an abrasion which has become septic, if this condition of sepsis is sufficient to produce suppuration an ischio-rectal abscess will probably result. In proof of this let me for a moment enumerate the signs and symptoms of what is known as a blind internal fistula. Intense pain is the first symptom of which the patient complains. If, now, during this period an examination is made, it will be found that there is a somewhat tense swelling of variable size underneath the mucous membrane situated between the two sphincters, possibly extending above the internal one. The

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swelling is due to a small collection of pus, the tension of which causes the severe pain. If this is left alone a temporary relief is very frequently obtained by the escape of some of the retained pus, but sooner or later the abscess cavity refills, and pain and swelling return worse than before. This repetition of events goes on until the abscess opens into the ischio-rectal region, and when this occurs there is for a short time a certain amount of relief. Soon, on account of the accumulation of the rapidly increasing pus, tension becomes even in this extensive area so great as to

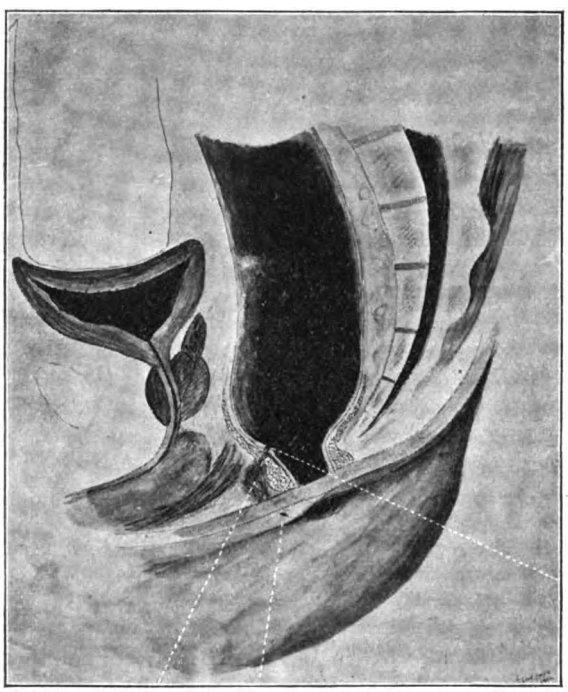

FIG. 5.—A. Internal opening. B. Track of fistula. C. External opening.

cause again considerable suffering. This is what happens when a blind internal fistula takes its own course. There are other ends to this condition which I will discuss later, but I have merely taken this as an example to endeavour to illustrate my statement that a complete fistula is always preceded by an ischio-rectal abscess, and that the origin of the abscess is this often-mentioned ulcer, which becomes septic. Bearing this in mind, the usual sequence of events is as follow :—The ulcer becomes septic, invasion of the tissues by the streptococci and the *Bacterium coli commune* progresses until the ischio-rectal fossa is invaded; this rapidly becomes distended, and then shows the signs of an ischio-rectal abscess, which in due course is opened; the retained pus escapes; the inflammatory condition settles down, leaving a track which is the fistula.

The Diagnosis and Treatment of Fistulæ when not Tubercular in Origin.—The diagnosis of fistula is not a matter of any difficulty, especially when the fistula is a complete one; that is to say, when there is a well-marked internal opening with a track leading to the external opening. Here the external opening will be recognised, and on palpation a definite track of inflammation will be found to extend towards the bowel. This has quite aptly been likened to the feeling of the stem of a clay pipe. As to the position of the internal opening, it will be remembered that the original internal opening is always situated between the sphincters.

The varieties of fistulæ are the internal, which are usually submucous, but may and frequently do extend beyond the submucous tissue; and the other and common variety is the complete fistula which has already been described. I do not recognise the blind external fistula, so-called, believing as I do that all fistulæ originate from within the bowel. Even supposing that no internal opening can be detected, that is no proof that none has existed. The other varieties not uncommon are those which are known as horseshoe or semi-horseshoe fistulæ, and in these conditions the suppuration has tracked round the external margin of the sphincter, either posteriorly or anteriorly (more frequently the former), to a greater or less extent. These fistulæ have one or more external openings, but usually only one internal opening—a further suggestion as to the origin of this condition. The treatment of a fistula is a simple matter. There are one or two points in regard to it which must be carefully attended to. In the ordinary, straightforward fistula, which is merely a straight track running from the external to the internal opening, all that is necessary is to pass in a probe-pointed director along the track, bringing the point out at the internal opening, and then further pushing it until the point of the director can be brought outside the bowel again, thus exposing the whole track outside the bowel.

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A curved sharp-pointed bistoury is passed along the director, and the superjacent structures are divided. Careful search is then made for any further burrowings or tracks which may exist, and these must be laid open. The hæmorrhage, which is at times quite sharp, is nearly always checked by packing, and this packing is a matter of some considerable importance to the eventual curing of the patient. Another point which must be carefully looked to is that no prolapsing piece of mucous membrane or skin shall fall down into the incision made. Should this occur it must be removed, otherwise healing will be considerably retarded. At the risk of being tedious I will again repeat that the original internal openings are always situated between the two sphincters. Occasionally there is another opening higher up, which may exist some inches above the internal sphincter, and up to which a director will readily pass; but in all these cases this high opening is a secondary matter, and not the original opening. It is a serious question what should be done under these circumstances, for if all the intervening tissues between the external and this high-placed internal opening be divided, necessarily both sphincters will be divided as well as some portion of the levator ani. The result of this is temporary incontinence at all events, and possibly this lamentable condition may become permanent. Moreover the whole of this divided area must granulate up, and the ultimate fibrous-tissue contraction produces constriction if not a stricture, and this in addition to any possibility of fæcal incontinence. It seems to me, therefore, that every and any method should be tried before this is taken into serious consideration, if, indeed, it is ever desirable to do so. My own practice has been always to avoid dividing these high-lying internal openings, and I do not hesitate to say that in these cases free incision externally, accompanied by the division of the original tract, the internal opening of which will be found between the sphincters, and the careful and systematic packing of whatever cavity or track leads up to the second and high-lying internal opening will eventually be found to prove effective. It may be that the treatment in a case of this kind will extend over many months, but if the ultimate result of cure is obtained without dividing the internal sphincter, the time will have been well spent.

The following is an example of this.

A patient weighing 22 st., and proportionately big, came to me with a large ischio-rectal abscess. When this was opened a track was found, which ran up to the bowel, seven inches in length. The original internal opening was between the sphincters, and this was laid open, the incision being extended freely out on the buttock; the high-lying track was packed. The rest of the wound healed, and the track was treated for a little over twelve months, when it healed, and has now remained perfectly sound for nearly three years.

The horseshoe variety of fistula is treated very much on the same lines as the one which has just been described. The direction of the track can be made out by palpation, and when the fistula is laid open the whole of this tract must be treated by free incision, scraping, and careful packing. It is desirable to only divide the external sphincter in one place. Occasionally in horseshoe fistulæ there are two separate openings between the sphincters, but only the worst of these should be laid open to start with, as the double division of this muscle causes often permanent weakness as to its controlling power.

Internal Fistulæ.

These have merely tracked underneath the mucous membrane and have not yet invaded the ischio-rectal fossa; and are treated best by first fully dilating the external sphincter and then dividing the invaded portion of the mucous membrane. But in cases where the track passes underneath the internal sphincter it is better that this should be left, and that a free incision should be made outwards from the internal opening, through the external sphincter and surrounding tissue. These cases of internal fistulæ are quite difficult to deal with, especially where the condition has existed for long and the surrounding tissues have suffered from inflammatory invasion. The after-treatment is simple enough, the sometimes extensive wound must be kept thoroughly clean and carefully packed at least once a day; but when possible the dressing should be applied twice a day. In addition, a daily warm bath, in which the patient sits for twenty minutes or more, is a great help in the healing of these cases. There is one thing to beware of in the treatment of these fistulæ, especially when, from the size of the

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wound, the granulating process is a long one. It is what is known as "bridging." By this is meant that, owing to some portion of the granulation tissue breaking down and the layer of tissue above remaining sound, a cavity is left, and all the rest of the wound may heal up with the exception of this broken-down track; the result being that the patient's condition is exactly as it was before the operation commenced, and the whole wound must be opened up again. In order to avoid this the packing should be always carefully and thoroughly applied, and careful probing and pressure should be made, especially after the first ten days, to discover whether the probe will pass into any bridged-over cavity, or whether any pus existing in a cavity can be pressed out. If such is discovered the overlying granulations are easily broken down with a stout probe or cut with scissors, and packing is then adjusted to the bridged-over cavity. This most essential precaution should always be carefully undertaken. One other point in the after-treatment of these cases is the form of dressing which should be used. It will be found, after a certain number of days, that the wound gets tired, so to speak, of any particular lotion or dressing, and does not respond to it as when it was first applied. Therefore, the changes are rung between two or three different applications. Peroxide of hydrogen, red lotion, chinosol, solutions of mercury, are all good in their way and for a time as lotions. Iodoform gauze, cyanide gauze, and plain lint soaked in one of the aforementioned lotions are also useful. It may be necessary occasionally to stimulate granulations by applications of nitrate of silver.

This concludes for the time being those disorders which I have stated owe their origin to the ulcer situated between the two sphincters, and those who have followed out the reason which I have given for so doing will, I think, admit that there is at all events a fair amount of fact upon which to build this suggestion.

Fistula due to Tubercle.

The diagnosis and onset of fistula when due to tubercle is quite different to that condition of fistula of which I have already spoken, and which is invariably prefaced by an acute abscess. Tuberculous fistulæ commence insidiously, and often, in fact generally, are not brought to the attention of the surgeon until the ulceration has considerably advanced. The condition is much more frequent in men, and the general appearance of the patients is one which we commonly recognise as usual in tuberculous constitutions. The local appearances show considerable undermining of the skin, which has around the affected area a livid appearance. There is little or no induration. The internal opening is usually large and ragged and ulcerated. The lungs of these patients are nearly always affected, and no treatment should be undertaken until a definite opinion has been given by a physician upon the state of activity of the lung trouble. Various opinions have from time to time been expressed as to the treatment of these patients. On the whole, the opinion of physicians in the past has been against any operation being performed for the local relief of these sufferers, and this has been based on the grounds that the lung trouble waxes and wanes in inverse ratio to the condition of the fistula, and that if the fistula is healed the activity in the progress of the disease in the lung is considerably increased. This is a general statement which is made, but upon what grounds I have not yet been able to ascertain. That a progressive ulceration, very slow in character, in this particular part of the body should have any effect except a bad one upon the general condition of the patient, and thereby affecting the local lung condition, seems to me unreasonable to suppose; and many instances might be given where operations have been carried out in other parts of the body in a phthisical subject without any bad effect appearing. It is only reasonable to suppose that no surgeon would undertake any operation upon an individual who was suffering from progressive phthisis, or whose condition was such that any operation might start the lung affection into greater activity. But after all this question will, I suppose, always be considered from one's particular experience and ideas, after carefully considering what others have said or done.

The first essential to my mind in regard to the question of operation is that no general anæsthetic shall be given. Various instances, with some of which we are most of us probably familiar, have been recorded where acute lung mischief has been set up after more or less prolonged general anæsthesia when operating upon a patient suffering from phthisis. The next important point is that

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the patients shall not lie up, but after a short interval of rest they shall get about. These two general principles I have always acted upon in dealing with tubercular fistulæ, and I cannot recall that anything but good has been the result of the treatment. The local treatment consists of injecting eucaine subcutaneously, laying open the fistulous tract, and scraping away any broken-down tissue that exists. Another application on the surface thus treated is made with eucaine, and then either pure carbolic or pure lactic acid is applied freely over the whole area. After two or three minutes' exposure to the air the tissues, which at first turn greyish black in colour, soon assume a vascular appearance. Care is taken that none of this strong acid trickles on to the surrounding healthy skin. The part thus treated is packed with sterilised cotton wool, and is kept as dry as possible. Wet applications are not desirable in these conditions, where vitality is already sufficiently low, and any such application is apt to make the tissues œdematous and still more feeble.

The after-treatment consists of cleanliness, daily packing, and an occasional application of lactic acid over the affected part. The general condition of the patient is looked after, and seaside air when possible should be insisted upon. With this treatment these patients slowly but surely improve, and, after often a considerable number of weeks, the fistula heals and remains sound. When this has been achieved it will be found that the general condition of the patient has materially improved.

Cancer and Malarial Infection.—To those who have caught at the straw of an antagonistic action between malaria and cancer, it will be somewhat depressing to learn of Prochnik's observations in the Dutch East Indies ('Wiener klinische Wochenschrift,' 1902, No. 5; 'Berliner klinische Wochenschrift,' June 2nd), to the effect that malarial infection is neither curative of carcinoma nor protective against it. It seems that cancer of the liver is very common in comparatively young persons in that part of the world, and that the fact is attributed to the frequency with which cirrhosis of the liver occurs, an affection thought by many distinguished tropical physicians to be a very frequent sequela of malarial disease. Apparently, therefore, malarial infection actually favours the development of cancer.—*New York Med. Journ.*, July 12th, 1902.

A POST-GRADUATE LECTURE

ON

BANTI'S DISEASE AND ITS TREATMENT.

Delivered at Westminster Hospital, June 24th, 1902.

By WILLIAM MURRELL, M.D., F.R.C.P.,

Physician to the Hospital, and Clinical Lecturer on Medicine and Joint Lecturer on the Principles and Practice of Medicine at the Hospital.

SOME difference of opinion exists as to the exact meaning which should be attached to the term "Banti's disease." By most writers it is considered to be synonymous with "splenic anæmia," "splenic cachexia," "splenic pseudo-leukæmia," "lymphadenoma splenicum," and the "splénomégalie primitive" of Bruhl. Others regard it as a late stage of splenic anæmia, and consider that fibrosis of the spleen and cirrhosis of the liver are essential factors in its diagnosis. On reference to Banti's original papers * I find not a word about cirrhosis of the liver, but he speaks of tumefaction of the liver and spleen and of idiopathic hypertrophy of those organs. He regards it as a direct result of the splenic affection, and not, in the inverse sense, of the splenic enlargement due to the cirrhosis of the liver; it is, he says, a true hypersplenomegaly. The essential features of the disease, as described by Banti, are: (1) a progressive idiopathic anæmia; (2) grave disturbance of the organic functions; (3) irregular fever; (4) hæmorrhages; (5) a fatal termination.

It may be a toxæmia, the point of entrance of the poison being the intestines, but on this point bacteriological examination throws no light.

Clinically it belongs to the group of the blood diseases. These may be conveniently divided into two categories: (*a*) the anæmic, and (*b*) the hæmorrhagic. In the first division we have (1) the primary anæmias, chlorosis, pernicious or essential anæmia, splenic anæmia, and general lymphadenoma or Hodgkin's disease; (2) secondary anæmia; and (3) leucocythæmia, including the spleno-medullary and lymphatic forms. In the second division we have (1) purpura, including

* 'Contribuzione allo Studio dell' Anemia Progressivo,' Firenze, 1881; and "Dell' Anemia Splenica," 'Archivio di Senola d'Anatomia Patologica,' 1883.

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the neurotic, arthritic, and malignant forms; (2) scorbutus of the sea and land varieties, and infantile scurvy; (3) hæmoglobinuria; (4) hæmophilia, or the hæmorrhagic diathesis; and, perhaps, (5) lipæmia. Splenic anæmia clearly falls into the first of these categories, and is most closely allied to pernicious anæmia. An examination of the blood bears out this contention. Taking the normal number of erythrocytes as 5,000,000 for a man and 4,500,000 for a woman per cmm., we find that there is a considerable falling off in this respect. The average blood-count in fourteen cases of Banti's disease was 3,336,357 erythrocytes per cmm. In Banti's three cases the numbers were 3,999,000, 3,948,000, and 2,851,350 respectively. Hayem considers that when in any disease the number of red blood-corpuscles falls to 1,000,000 the patient is in extreme danger, and that life cannot be sustained if they are reduced to 300,000 or even 400,000. In one of my cases there were only 362,000 erythrocytes per cmm.; and this is not the smallest number published, for Quincke's record is 143,000 per cmm. Not only is there no leucocytosis, but the white blood-corpuscles are much reduced in number. In one of my cases instead of 7000 there were only 4375 per cmm., the hæmoglobin value being 20 per cent.

Next to the changes in the blood-condition the alteration in the spleen and liver are most marked. The spleen in one of my cases weighed nine ounces, was dark red in colour, beefy, and homogeneous. On microscopical examination the connective-tissue framework was found to be much thickened. During life attacks of pain in the region of this organ are not uncommon, and are due to perisplenitis, a condition frequently met with in splenic leukæmia. The liver is often slightly cirrhosed and much pigmented, and the cells are misshapen, atrophied, and granular. This, however, is not always the case, and the liver is sometimes normal in appearance, and shows nothing abnormal when examined microscopically. Attacks of hæmorrhage, as is common in many blood diseases, are of frequent occurrence, and may assume the form of hæmoptysis, hæmatemesis with melæna, epistaxis, obstinate bleeding from the gums, or purpura. In a case of acute Banti's disease recently under my care at the hospital the patient brought up three pints of dark clotted blood, but at the autopsy made a few days later there was no trace of gastric ulcer or of any lesion of the mucous membrane of the stomach. Such cases are extremely deceptive, for unless an examination of the blood is made they are easily mistaken for acute gastric ulcer, cancer of the stomach, or ulcus carcinomatosum. An examination of the vomited matter and the discovery of the Oppler-Boas bacillus would be of diagnostic importance, and would point to gastric carcinoma. It is said that in unstained preparations these organisms are readily recognised by their large size and peculiar baseball-bat shape. They possess the property of forming lactic acid from various kinds of sugar, a constant parallelism being observed between the occurence of these bacilli in large numbers and an excessive formation of this acid. Kaufmann demonstrated their presence in the vomited matter in nineteen out of twenty cases of gastric carcinoma. Schlesinger and Kaufmann state that although there are many other organisms which form lactic acid their presence is suggestive. Hemmeter attaches much importance to their discovery, but they are not always present, for in a case of malignant disease of the pylorus recently under my care, the diagnosis being confirmed by an autopsy, nothing corresponding to the particular organism could be detected, although careful search was made.

The skin diseases and complications met with in Banti's disease are of the hæmorrhagic type, such as petechiæ, subcutaneous hæmorrhages, ecchymoses, and melanodermia, and are similar to those found in lymphatic leukæmia. The course of the temperature is of an irregular hectic type.

The diagnosis, to any one familiar with the essential points of the disease, presents no difficulty, but is necessary to distinguish it from simple pernicious anæmia, leucocythæmia, Hodgkin's disease, and alcoholic cirrhosis of the liver. When the spleen is exceptionally large it may be confounded with syphilitic or tuberculous disease or abscess of that organ, and perhaps with pernicious ague. It is probable that splenic anæmia is by no means an uncommon disease, but that it is frequently overlooked, for there are not more than forty published cases, all told.

The prognosis is bad, and nearly all cases sooner or later terminate fatally. I have known a case last two years, and on the other hand I have

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known death ensue in sixteen days from the onset of the very first symptom. Much, however, depends on the mode of treatment adopted, and in this respect there has been a notable improvement of late.

With regard to drugs, it must be admitted that iron, which is so useful in chlorosis, is of very little value. A greater measure of success, however, attends the exhibition of that group of remedies which includes arsenic, phosphorus, and vanadium. Most of our cases, before admission to the hospital, have been treated with arsenic, and the skin, of the abdomen especially, is often deeply pigmented. This sometimes leads to a suspicion that the adrenals are affected; but this is not really the case, and at the autopsy these organs are found to be healthy. Vanadium is closely allied in pharmacological action to phosphorus, and given in large doses to animals, produces fatty degeneration of the liver. The salt commonly employed is the meta-vanadate of sodium. It is freely soluble in water, and the dose is 2 milligrammes daily. It has of late been employed in various forms of anæmia, and apparently with some success.

The surgical treatment of cases of Banti's disease calls for some comment. Splenectomy has been performed by Maraglioni and others, and although the operation is not unattended with danger, in carefully selected cases the results are favourable. Splenopexie has, I think, little to recommend it. The mode of procedure which appeals most to me is that which aims at the establishment of a collateral circulation between the portal and systemic systems, especially in cases in which ascites is a prominent symptom. This operation has proved strikingly successful in the treatment of various forms of cirrhosis of the liver. It was originally suggested by Talma, of Utrecht, in 1887, and in 1891 Lens, of Paramarebo, operated on a negro suffering from "cirrhosis hepatis atrophica." The patient recovered from the effects of the operation, and died some six months later, apparently from general weakness. At the autopsy it was found that the blood-vessels were greatly increased in size, and that "the omentum was thick and rich in blood." The account of this case, which was published in Dutch, attracted but little attention. In 1896 Dr. David Drummond, of Newcastle-on-Tyne, hit on the same idea, and several of his patients were operated on successfully by his colleague, Mr. Rutherford Morison. Since then the operation has been largely practised both on the Continent and in America. I have published* a historical *résumé* of the subject, with an abstract of the leading cases.

The first occasion in which this mode of procedure was applied to Banti's disease was in 1899, when Mr. Walter Spencer, at the Westminster Hospital, operated on a case under my care. The patient was a girl æt. 19 who presented all the typical symptoms of splenic anæmia. Her blood was examined on many occasions, and her red corpuscles usually numbered about 2,700,000 per cmm., and the leucocytes 3360. In addition to the anæmia she had an enlarged spleen, a small cirrhotic liver, and ascites. An incision one and a half inches in length was made halfway between the umbilicus and the pubes, and a large glass tube was introduced, which served to draw off some eight pints of blood-stained serous fluid. A second incision of about the same length was then made in the median line above the umbilicus, and a piece of the omentum two inches square was attached by kangaroo stitches to the anterior abdominal wall. There was no shock, and the patient made a good recovery. Three weeks later the superficial abdominal veins were larger, and the wound was practically healed. There was no return of the ascites, and the patient was discharged much improved. Four months after the operation she returned, and stated that until six weeks previously she had been perfectly well, and had been able to attend to her duties. It was considered inadvisable to repeat the operation, and a month later she died. At the autopsy firm adhesions were found between the omentum and the abdominal wall, and there were organised adhesions between the stomach and the diaphragm, and between the liver, spleen, and diaphragm.

Although in these cases it is necessary for the physician to enlist the co-operation of a surgical colleague, much of the success of the treatment depends on the medical treatment subsequently adopted. The bowels should be kept open by the administration of a hydragogue purgative. I do not find that jalap is well suited for this purpose,

* 'Medical Press and Circular,' January 8th, 1902; 'Lancet,' June 7th, 1902.

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but I have obtained excellent results from the administration of Turpethum or Turpeth, the dried root and stem of the *Ipomœa turpethum*, a drug introduced into the Indian and Colonial Addendum (1900) of the 'British Pharmacopœia.' The powder has a faint odour and a somewhat disagreeable taste. The dose is said to be from five to twenty grains, but I find that fifteen grains or more is required to produce a free action. I am satisfied that the enlargement of the superficial abdominal veins is greatly promoted by the use of counter-irritants. Blisters are not suitable for the purpose, but an ointment made with capsicum (12 grammes), spermaceti (6 grammes), and olive oil (44 grammes) answers admirably. Another useful external remedy is the Oleum cajuputi. In addition to acting as a counter-irritant it is absorbed and gives relief to the flatulence which is often so distressing a symptom in these cases. When there is much pain from perisplenitis or perihepatitis I order a chloroform poultice made by sprinkling a teaspoonful of chloroform on several thicknesses of lint, and apply it to the abdomen. It gives rise to a good deal of smarting, but if the patient is instructed to shift it about when the pain becomes unbearable the effect is by no means unpleasant.

Lord has collected twenty-six cases of typhoid spine, and has analysed them. Of the twenty-six, twenty-two occurred in men. In six mechanical strain is given as a cause beside typhoid fever. In fourteen cases the pain began during typhoid or within two weeks afterwards; in eight cases it occurred within six weeks; in the remaining four, in three months. There was tenderness in sixteen, and pain in all twenty-six. Stiffness of the spinal column was noted in eight cases. In six cases only was hysteria or neurasthenia observed. Kyphosis occurred in eight cases. Rest in a plaster jacket is the best treatment. The prognosis is good. The condition is generally perispondylitis or spondylitis. A rise of temperature in seven cases was suggestive of organic trouble.—*Philadelphia Med. Journ.*, July 5th.

Messrs. Cassell and Company have in the press a new and revised edition of Dr. Burney Yeo's popular 'Manual of Medical Treatment,' upon the preparation of which the author has been for some time engaged. This new edition, it is expected, will be ready about the end of September.

WITH DR. DONALD HOOD

IN THE WARDS OF

THE WEST LONDON HOSPITAL,

JUNE 30th, 1902.

Mitral Stenosis.

Our first patient is a stonemason æt. 41. He was admitted about ten days ago for dyspnœa, a dyspnœa which occurred on movement. As he lay in bed there was but little if any disturbance of respiration. He was a high-coloured man with a very irregular, small, and thready pulse. There was a congested state of his veins. You see his jugular veins stand out now; they did so far more when he first came in, and there was also very marked pulsation. We found, on examining his cardiac area, that there was dulness extending far over to the right side, in fact there was no disconnection between the liver and the cardiac dulness. To-day you will observe that the dulness is not quite so evident. The liver was very much enlarged, and was three or four inches below the ribs. His apex beat, as far as one could gather, was as you feel it now, in the fifth space just outside the nipple line. On auscultation no bruit could he heard. Both the first and second sounds were short. I examined him carefully on three occasions, but could detect no bruit. It is evident that all the trouble is referable to the right side of the heart. Now, there is nothing in the chest to account for the cardiac change. There is no history of chronic bronchitis, nor of asthma. I believe the most ordinary cause for such a condition as we have here before us to be stenosis of the mitral valve. The absence of a bruit does not negative contraction of that valve. In some of the most advanced forms of this disease there is no bruit. About twelve months ago we had a case of a similar nature, but perhaps in a later stage. The conditions of the heart were much the same as we see here, we were able to verify the diagnosis of mitral stenosis. The mitral valve would admit of only the passage of a penholder. But the patient, while under observation, never had any bruit. There are many cases of mitral stenosis in which, if you see them in what one might perhaps

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call the congestive stage, that stage in which there is congestion of the portal and pulmonary system, no bruit may be present, but as the equilibrium of the circulation is restored a bruit will develop. My house physician tells me that he heard a slight pre-systolic murmur in the patient whose case we are now considering. If so, I think it renders the diagnosis of disease of the mitral orifice as established.

Since his admission the patient has made great progress. He is better in every particular, and it is possible that as he improves a less pronounced pre-systolic murmur will assert itself. His pulse is better sustained, and there is a greater volume of blood passing from the left ventricle. With regard to treatment, it is no use giving such a case cardiac tonics; in fact, they bear digitalis badly. I have thought this point has often helped our diagnosis. Here, the patient was ordered digitalis on his admission, but he derived no benefit. The left ventricle is quite strong enough for all the work it has to do. If you give digitalis you increase power where it is not wanted. You are apt to increase the local congestion by stimulating the right ventricle already working to the breaking point. The irritability and cardiac distress are best met by unloading the portal system by purgation. I have ordered blue pill and compound rhubarb with a morning saline draught. The patient is also taking a mixture containing five drops of strophanthus and a few grains of ammonia citrate of iron. Under this treatment he has made rapid improvement.

Syphilitic Laryngitis.

This man was admitted for spasmodic dyspnœa. The trouble was evidently laryngeal, and it was a question whether tracheotomy might not be immediately required. He had a very distinct history of syphilis, and we concluded that he was suffering from syphilitic disease of the larynx. Dr. Ball examined the larynx and verified the fact; he also detected disease of the posterior nares, which was probably of a syphilitic nature. Since his admission he has been under the influence of fifteen grains of iodide of potassium three times a day, with a drachm of the solution of bichloride of mercury, and now he is in a very different state. He is able to breathe freely with no difficulty, and there is no dyspnœa. During the first forty-eight hours after admission he had several severe attacks of spasmodic dyspnœa which were very urgent.

Our next patient is a man æt. 60, who came in with a history that he had been jaundiced six weeks. He also had general debility. I found his liver enlarged, especially to the left, slightly nodular, and over the region of the umbilicus there was a distinct infiltration, which is an important symptom, and its significance was, I believe, first pointed out by the late Sir William Jenner. The favourable points in his case are that there has been some slight decrease in the jaundice. There is more bile passing by the stools, and there is less bile in the urine. There are no gastric symptoms, such as vomiting or loathing of food. I thought it right to try the effect of iodides, and he is taking ten grains of iodide of potassium three times a day; but I am afraid that the disease is malignant. In obscure disease of the liver it is best to reserve one's judgment and watch the course of events. Malaria abscess, gallstones, syphilis, and malignant disease all come before us under such varying and manifold forms that in any individual case with ambiguous conflicting symptoms it may be impossible to pronounce on the true cause of the disease at first sight.

Simple Lobar Pneumonia.

The next patient is a case of simple lobar pneumonia, the crisis of which occurred on the second day following admission. There is nothing particular to call your attention to beyond the fact that the disease has run a typical course. The moist sounds now to be heard over the affected area are those of resolving pneumonia. During the past few weeks we have admitted a large number of cases of acute lobar pneumonia. With but few exceptions they have all run a simple uncomplicated course. How strikingly different from the effects of pneumonia as seen during the presence of influenza! During an influenza epidemic cases of pneumonia succumb case after case, and I have a strong feeling that the pneumonia at those times is grafted with the poison of the influenza germ. Moreover, the patients during an influenza visitation so soon lose power. Their vitality is materially influenced. In the treatment of ordinary cases of lobar pneumonia

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practically all one has to do is to carefully watch the patient and guide him past the rocks, but during an influenza epidemic you will require more stimulants, and you must keenly observe every untoward sign.

ADVANCED AORTIC REGURGITATION.

The next man has been in the ward many weeks with aortic regurgitation accompanied by great weakness. He is much better than he was, and I attribute a good deal of his comfort and his improved condition to the influence of opium. He has had opium for several weeks, ten grains during the twenty-four hours. Previous to the use of the drug his discomfort was great—cardiac pain, restlessness, dyspnœa, and insomnia. The opium has not caused any inconvenience. There is no constipation or loss of appetite. Opium is one of our most valuable remedies in the treatment of organic disease of the heart.

Cheese Poisoning (Tyrotoxismus).—The report of Dr. Collingridge, Medical Officer of Health for the City of London, contains the following account of an interesting series of outbreaks of poisoning from cheese which have taken place in various parts of London during the year, and which have been all traced to one common source. Though, so far as is at present known, no cases have occurred within the City limits, it is an undoubted fact that within the City's jurisdiction the cheese causing the trouble was in the first place sold by the importer to two large firms, and again resold by them to various retail shopkeepers. From the one factory through the same shipper were obtained cheeses made about the same date, which, being sold in Bethnal Green, Bermondsey, Croydon, Deptford, Finsbury, and Tunbridge Wells, their consumption was followed by illness in those who ate them. It thus appears that there was a definite causal relation between this particular consignment of cheese and the various outbreaks. It was not possible to separate or identify the "batches" or "vats" of cheese, and the shipping mark in no way assists one in this respect. Assuming that some amount of poisonous material was present, it might be in small quantities, and in all probability would be very unequally distributed throughout the consignment. The maker of the cheese failed to detect anything unusual in the appearance of the cheese when ripened ready for shipment. The fact that the cheese did not produce symptoms in all cases when eaten would further tend to show that the poison was not present in all portions. From time to time, doubtless, such cases occur, and the chief interest in the present outbreak is the fact that it was possible to trace them all to the same shipper, and to a shipment about the same date, though of different marks. The symptoms in all the above cases were similar, and are those usually ascribed to the action of tyrotoxicon or tyrotoxin, a ptomain which is sometimes found in poisonous cheese. This is one of a large class of substances formed by the action of bacteria upon nitrogenous compounds, which in the majority of cases produce well-marked symptoms. It is an exceedingly unstable body, being easily destroyed by the action of heat and moisture, and even by exposure to the air; and being present in very small proportions, it has never been isolated in sufficiently large quantities to allow of its composition being definitely determined. Its chemical and clinical actions are precisely identical with those of diazo-benzol, which is simply a transition product of putrefaction. Most of the observations upon this material so far have been made by Professor Victor C. Vaughan, of Ann Arbor, Michigan, who has had the opportunity of investigating several outbreaks caused by it. It appears probable that it occurs in the milk, and it has been suggested as one of the causes of the wide-spread outbreaks of summer diarrhœa among children. Professor Vaughan mentions two outbreaks in Michigan in 1895, when fifty persons were affected by cream, and about twelve by cheese. The toxicogenic germ found was similar in both outbreaks. It grows readily on the ordinary culture media, and also many fruits and vegetables. This latter point is of great interest, as it is well known that milk often produces unpleasant symptoms if it has been kept near unsound and decomposing fruits and vegetables. Although the symptoms are very marked, the result is practically never a fatal one in adults, though a patient may be left in a weak state for some considerable time. Practically the difficulty is that in the manufacture, as the cause is not known, it is impossible to take any steps to prevent the formation of this ptomain; and further, as it imparts no taste nor smell to the cheese, its presence cannot be detected except by analysis.

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A LECTURE
ON
THE ACTION OF MUSCLES.

Delivered at the National Hospital for the Paralysed and Epileptic, Queen Square, London, July 1st, 1902.

By C. E. BEEVOR, M.D., F.R.C.P.,
Physician to the Hospital.

GENTLEMEN,—I propose to say a few words this afternoon about the action of muscles, and then to show you some cases in illustration of some of the points referred to.

In regard to action of muscles, the first thing to determine about them is, how they act. What is the best way of ascertaining how a muscle acts? There are three chief ways. There is the anatomical way, in which one dissects the muscle out and pulls upon it and observes what it does to the joint. In the case of the biceps you see there is a certain action upon the radius. The next is the method employed by Duchenne, namely, the electrical method, by which one faradises a muscle and then notices what happens to the joint over which it is inserted. An objection to both these methods is that you simply take one muscle and presuppose that one muscle can act by itself in performing movement; and although a muscle may perform a certain movement when pulled upon, one does not know whether the muscle enters into that movement in actual life.

With regard to the first point, probably there is no movement of the body in which one muscle acts by itself. It is perfectly impossible for the will to put into action any one muscle alone;* for instance, you cannot contract the biceps by itself, nor the tibialis anticus by itself. The will has actually no power over single muscles. All the brain does is to send down a message for the performance of certain movements. As Dr. Jackson

* Except in the case where a movement is produced by only one muscle.

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said years ago, the brain knows nothing about muscles; it only knows about movements. Therefore, it is impossible for the will to throw any single muscle into action. So that the anatomical methods employed in the dissecting-room are not really applicable practically. The same may be said with regard to the electrical method,—the throwing into action of a certain muscle and deducing its behaviour in life from that. To my mind the only true way is to reverse the process; that is to say, to take some movement and ask the person to perform it, and then see what muscles take part in this movement. For instance, you ask a person to flex the elbow, and then you see what muscles take part in that flexion. By this means you will ascertain that several muscles take part in the movement, and not only one. That is what we have to find out in every movement.

If one does that, one finds that for every movement there is a definite number of muscles which take part in it, and it seems to be the fact that the individual muscles which take part in this movement cannot be left out. You cannot by means of the will leave out certain muscles which are engaged in the performance of certain movements, and you cannot diminish the number of such muscles. The question is whether you can supplement them. It is difficult to say for certain, and very hard to prove. That is to say, if certain muscles in the group are paralysed, it is difficult to say whether other muscles can come in and take their place. A short time ago I held strongly that a muscle cannot be supplemented, but I am not prepared to say now that by education it is not possible to bring another muscle in to take the place of those which are paralysed. I will show you presently a man who has almost lost the power of flexing the elbow. He has lost the power of his biceps, his supinator longus, and his brachialis anticus. He flexes his elbow first of all by pronation of the forearm, and apparently the muscles of the forearm do take on the part of the flexors. I am not sure in that case whether all his work is not done by the pronator radii teres, which is one of the flexors for the elbow-joint. Therefore, there is a minimum list of muscles which you cannot diminish, and which may be looked upon as the ultimate components of a movement. And this inability to omit a member of a group is well shown by certain groups of muscles which always act together. You cannot do what I am doing, viz. extending the thumb, without putting into action the extensor carpi ulnaris and the flexor carpi ulnaris, no matter what the position of the hand is. If you extend your thumb you must bring out those two muscles, and it is impossible for the thumb to extend without at the same time bringing into action the extensor carpi ulnaris and the flexor carpi ulnaris. Why they come into action I will mention later. Another example is the action of the extensors of the fingers and the flexors of the wrist. We shall see presently in a case that if the extensors of the fingers are paralysed, then the flexors of the wrist overact, and there results a movement which is not intended—in place of extension of the fingers there is flexion of the wrist, because the brain does not know that the extensors of the fingers are gone. A message is sent down to move these muscles, the extensors of the fingers, which are the direct acting muscles; but they are gone, and the other muscles which take part in the movement act, and there is produced a different movement, and one which the patient does not want to perform; that is to say, you get flexion of the wrist and not extension of the fingers. Another illustration is afforded by the flexors of the fingers and the extensors of the wrist. If you flex the fingers and grasp, you throw into action the extensors of the wrist, as you can feel by putting your finger on the extensor carpi radialis. The two instances were pointed out by Duchenne, though I do not know whether he was the first to do so. There is another instance which I observed when I was working upon these muscles, namely, the question of flexion of the elbow-joint and that of pronation and supination of the forearm. The two chief muscles by which you supinate are the supinator brevis and the biceps. The supinator longus, I believe, has not a supinator action, though it is described as such; I believe it is a pure flexor of the elbow-joint. Anyhow, the two chief muscles in supination are the biceps and the supinator brevis. The biceps, as you know, is a very strong flexor of the elbow-joint,—in fact, it is one of the chief ones; but, as everybody knows, it is perfectly easy to supinate without flexing the elbow-joint. If you supinate your forearm, you can feel that your biceps is contracting strongly, but there is no tendency to flex the elbow-joint. It occurred to me that there must

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be something which is stopping it, which is preventing the elbow from flexing, and the most likely muscle would be the antagonist to flexion of the elbow, namely, the extensor—that is the triceps. If you take hold of an iron bar and supinate as hard as you can, you will find your triceps is contracting strongly, but as soon as you flex the elbow-joint the triceps leaves off. The same thing holds good, but less so, in the case of pronation. The pronators of the arm are the pronator radii teres and the pronator quadratus; but as the former is a slight flexor of the elbow-joint, when you pronate you also flex the elbow-joint. And there, again, the triceps steps in and prevents the elbow from being flexed. So you can pronate or supinate easily without any tendency to flexion of the elbow-joint, although some of the muscles which take part in these movements are powerful flexors of the elbow. One sees the want of this antagonism very well where the triceps is paralysed, and you ask the person to supinate the elbow flexes because there is nothing to keep the joint in a fixed position between flexion and extension.

The question is, what are those muscles which take part but are not the direct actors, in a movement, and what are we to call them? I believe it is still taught, as it certainly was, that in every muscular movement the antagonists take part; that is to say, when you flex your elbow the triceps also takes part in movement, to modify, or counteract, or alter, or regulate the movement of flexion. I will not go into the history of that belief, because it comes down from Winslow, I believe, and I think it is taught still. That is a theory as to antagonists which I cannot agree with. Some years ago I pointed out that in powerful movements the antagonists do not act, and you can prove this by extending your elbow against the table, when you will feel your biceps is quite flaccid. I pointed that out in a paper which I published in 'Brain' some years ago. Later, Professor Sherrington and Dr. Herring have shown experimentally that if they stimulate the motor area in an animal, and take as their point the part of the cortex where the representation of the extensor muscles of the elbow are localised, they find that not only does the biceps not take part in the action, but actually it is relaxed; that is to say, you get a negative action of the biceps. This proves that so far from the antagonist taking part in the extension of the elbow, it is relaxed. I think there is no doubt that the antagonists do not act.

Now, what are these extra muscles which have been described as taking part in the movements? If you look at them you will find that they are not antagonists; they are synergic muscles, that is to say, they act together in harmony with the prime movers of a joint. Take the flexors of the fingers and the extensors of the wrist in the moment of grasping, in which the former are the prime movers. What are antagonists? They are the muscles, I take it, which produce the movement which is diametrically opposite to that of the prime movers. For instance, in the case of the flexors of the fingers, the antagonists of them are the extensors of the fingers, not the extensors of the wrist. You cannot get from the extensors of the wrist a directly opposite movement to what you produce by the flexors of the fingers; therefore these muscles, which act together, are not antagonists, but they are synergics. That, I think, is one of the reasons why what I look upon as an error has crept in. The extensors of the wrists have been looked upon as being antagonists of the flexors of the fingers, but they are not so. Hence the antagonists do not act in the movement, but the synergics do, and the synergics are the muscles which do not produce the movement.

In flexing the fingers it is the flexors which are the primary movers; but other muscles are brought in for a certain purpose. What is that purpose? One may formulate it as follows:—If a muscle which takes part in a movement has two actions by passing over two joints, and if only one of those actions is required, an antagonist is brought into the group to neutralise the action which is not required. For instance, you use the flexors of the fingers which pass over the wrist-joint and the finger-joints in the act of grasping; if the muscle did its full work it would flex the wrist, because it has two actions—flexion of the wrist and flexion of the fingers. But you only want flexion of the fingers, and therefore, to prevent flexion of the wrist, you bring in its antagonist, which is the extensor of the wrist, and the remaining action is flexion of the fingers. Therefore, as the muscle has two movements, only one of which is wanted, you bring in the antagonist to counteract the movement which is not wanted. So with the movements of pronation and supina-

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tion. The muscles taking part in these movements have two actions, one to flex the elbow-joint and the other to pronate or supinate. You do not require flexion of the elbow-joint, and therefore you bring in the antagonist, namely, the triceps, which is the chief extensor of the elbow-joint, and you get pure supination or pronation without any flexion of the elbow-joint. Other examples occur through the whole body.

I said just now that the antagonists do not act. But there is one exception to this statement, namely, in the case of those movements in which the action of gravity takes part. In that case the antagonists alone act, and the muscles which are intended to produce the particular movement do not act. The simplest way of demonstrating this action is by standing up straight, and then leaning forward. In the erect posture, standing at "attention," if you place one hand on the back over the erectores spinæ muscle and the other over the recti abdominis in front you cannot feel any muscle contracting. But if you bend forward ever so little, you feel your erectores spinæ muscle stand out; or in other words, if you go in the direction of flexion of the spine it is not the flexors which act, but their antagonists, the extensors of the spine. Now that seems to be self-evident, and I should not mention it but for the fact that one reads in books that in bending the body forward in the erect posture it is the flexors of the spine which act. A moment's consideration will show you that if that were to happen, a person would be hurled to the ground with tremendous violence. What happens is, that whenever gravity comes in it is the antagonists which act and hold up the body and let it out by degrees, just as in the case of a weight being let out by a crane. A slight nodding of the head forwards is sufficient to bring out the erectores spinæ, and looking up to the ceiling suffices to bring out the recti abdominis. If you lean forward a little, the erector muscles come into action, and when you lean backwards the recti abdominis contract. In that way the erect posture is preserved. But it is always the antagonists which act in those circumstances, and not the prime movers. That is probably the reason why, in locomotor ataxy, the patients swerve so much when the eyes are shut. They do not receive intelligence quick enough as to the position of their body; and after the body has got too far towards falling the antagonist muscles contract and pull back the spine with a jerk; then the opposing muscle overdoes it, and consequently there is a swaying movement produced. I think it is probably due to the intelligence that the trunk has moved out of the perpendicular being conveyed too late for the muscle to act.

So the muscles should not be studied for each single muscle, but for their combined action in each movement. One muscle may enter into several movements, and we may have a muscle paralysed for one class of movements and not for another. This is probably only in cases where the lesion is in the higher centres, that is to say, somewhere higher up than the medulla or spinal cord. For instance, you may have the internal recti muscles of the eyes paralysed for the movement of convergence, and yet not paralysed for movements from side to side with the external recti. In the same way you may have the latissimus dorsi paralysed in hemiplegia when it acts as an arm muscle, but the same muscle will not be paralysed for acting as a bilateral muscle of expiration, and it can be made to contract by getting the patient to cough.

Another point is that, as far as I have seen, there is no muscle which takes a principal part in two different actions which are antagonistic to each other. It formerly used to be taught that the supinator longus was both a pronator and a supinator—that it brought the forearm into a midway position from either extreme pronation or supination. As far as I can see, that does not happen. I do not think it is either a supinator or a pronator; I look upon it as a pure flexor of the elbow-joint. The triceps certainly acts in both pronation and supination, but it comes in as a synergic, not as a principal muscle.

We have, therefore, a number of simple movements, the muscles entering into which cannot be left out, and the ultimate components of these groups can be known. The next question which arises is, Where are these groups localised? Are they localised in the spinal cord; or are they localised in the basal ganglia, or in the motor cortex itself, that is to say, the excitable area of the cortex, situated round the fissure of Rolando?

With regard to the localisation being in the spinal cord, I do not think that is likely; I think the spinal cord is only for reflex actions, and that

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the groups of muscles producing each movement are not represented in the grey matter of the spinal cord, for this reason: that you get sometimes two muscles which are inseparably connected with a definite movement, one of which may be paralysed in lesions of the spinal cord, and the other is not. Are these movements localised in the basal ganglia? I do not think so, because in cases of hemiplegia due to a lesion of the excitable cortex, these movements are all lost, whereas if the basal ganglia were the seat of their representation they would be preserved. I think it is most probable that the centres where these groups of muscles are represented are situated in the excitable so-called motor area of the cortex, the ascending frontal, and—to a less degree—the ascending parietal convolutions. In confirmation of that there are the experiments which Sir Victor Horsley and I made some years ago, in which we stimulated the cortex in the orang, and the results which we got were all single movements. Therefore it seems highly probable that the representation for these single simple movements is situated in the motor cortex. You get the same movement from stimulation of the internal capsule. So it is probable that the arrangement of these muscles for a definite simple movement takes place in the motor cortex, and that the messages are sent down to the anterior horns, but that the actual grouping takes place in the cortex. Complex movements are probably brought about by a rearrangement of the simple movements which takes place in other parts of the cortex, which are probably higher centres than the motor area.

Now, just to recapitulate, I should like to say that for each single movement there is probably a definite number of muscles taking part in it, the individual members of which cannot be omitted, and which may be regarded as the ultimate components of a movement. Secondly, if a muscle taking part in the movement have two actions, such as when passing over two joints, and only one is required, an antagonist is brought into the group to neutralise the action which is not required. Thirdly, the direct antagonists do not take part in the movement, but, on the contrary, they are relaxed, except in movements in the direction in which gravity acts, and then the antagonists alone contract, and gradually relax in the direction of movement. Fourthly, a muscle which takes part in two different movements may be paralysed for one and not for the other.

Then there is a last point which I omitted to mention, namely, that in hysteria you sometimes get an extraordinary condition. In hysterical paralysis it has been observed that when a patient is asked to perform a movement, uncertainty occurs, and this is followed by oscillation of the limb, and there is great difficulty in getting the movement carried out. I had a case under my care at the Great Northern Hospital, that of a girl in whom there was hysterical paralysis of the upper limb. When she was asked to flex the elbow, one noticed that the first movement took place in the extensors; that is to say, she did exactly the opposite movement she was asked to do. When she was asked to extend the elbow, the first movement took place in the flexors of the elbow. That was followed afterwards by a short oscillation between the two, displaying that to-and-fro movement which one notices in these cases. In hysterical paralysis, therefore, when the patient can perform some movement, the antagonists may act first, and are then followed by contraction of the muscles proper to the movement. Those are the only cases which I know of in which the antagonists act first in the performance of any movements, excepting those where gravity acts.

[Cases were then shown to illustrate the above points. In one case in which there was paralysis of the extensors of the fingers and of the wrist, it was shown that in grasping the wrist became strongly flexed, because one of the actions of the flexors of the fingers—that of flexing the wrist—which was not required was not neutralised by the action of the extensors of the wrist, which were here paralysed.

In another case, where there was paralysis of the biceps, brachialis anticus, and supinator longus, the patient, in flexing his elbow, first pronated the forearm by the action of the pronator radii teres, and this muscle then flexed the elbow-joint, and it was apparently helped by all the muscles passing over the wrist, but to a very slight degree, as it did not seem to make much difference to the strength of the movement whether the muscles acted or not.

Another case, of old infantile paralysis, was shown where the small hand muscles were preserved, and also the flexors of the fingers and the flexor carpi ulnaris, but the flexor carpi radialis and the extensors of the wrist were paralysed.]

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THREE CLINICAL LECTURES

ON

THE DIAGNOSIS AND TREATMENT OF RECTAL DISEASES.

Delivered at the Medical Graduates' College and Polyclinic, May 26th, June 2nd, and 9th, 1902.

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LECTURE II.

HÆMORRHOIDS.

PERHAPS the most common complaint of all those which occur in this portion of the body is hæmorrhoids, and it is rare for any one to escape some hæmorrhoidal condition during life. Certainly many have only slight symptoms, which are easily remedied, but a large proportion suffer sufficiently to require treatment. The principal symptom of hæmorrhoids is pain, at least that is what most patients complain of; but one which is quite as common and often more serious is hæmorrhage, which from its constancy may cause the most profound anæmia. The various kinds of hæmorrhoids may be simplified into external and internal. These have practically nothing to do with each other, one being dependent upon the blood extravasation in connection with the inferior hæmorrhoidal veins, and the other to do with the varicose condition of the superior hæmorrhoidal plexus.

To deal with the external hæmorrhoid first. This is usually quite sudden in origin, and consists of a painful, tense swelling on the muco-cutaneous margin. If left alone, after three or four days of by no means inconsiderable pain, the swelling subsides, and beyond a redundant fold of skin there is no harm done; but sometimes the external swelling becomes inflamed and suppurates, and it must then be treated like any other abscess. The treatment of an external hæmorrhoid is quite simple, and produces immediate relief. Either with or without a preliminary injection of eucaine the swelling is incised by means of a curved bistoury. When the incised parts are separated it will be seen that the swelling has been caused by blood-clot. This is removed, the œdematous skin over it is preferably cut away, and some simple *dry* dressing is applied. In two or three days this wound has healed, and the immediate effect is the disappearance of all pain, which in these cases at times is so severe that the patient is unable to sit down or walk, or lie for any length of time in any one position.

Internal hæmorrhoids, as I have already mentioned, have to do with the superior hæmorrhoidal plexus, which we have already located as situated at or about the level of the internal sphincter.

I would like here to call attention again to the circulation of this area.

Writers on this subject have given various kinds of hæmorrhoids, according to their shape, and also with regard to their attachments to those various folds or columns of mucous membrane which I have already referred to; but I do not think that any useful point will be gained in these lectures, which I am endeavouring to make as practical as possible, in discussing this question of variety. Suffice it that all hæmorrhoids give rise to the same symptoms, and mostly require the same treatment. The symptoms have been already mentioned, namely pain and loss of blood. The complications are prolapse and strangulation, and this latter, when not reduced, leads to gangrene, which may further end in a condition of septic ulceration—a serious condition, to be discussed later.

So many patients come to me with the statement that they are suffering from hæmorrhoids, and the statement is frequently made upon the assurance of their doctor, who without any examination has taken it for granted that the symptoms the patient complains of are necessarily due to hæmorrhoids. This mistake sometimes is of little importance, but at other times is quite a serious error. The three troubles which are commonly put down to hæmorrhoids are: (1) pruritus ani, (2) fissure, (3) carcinoma. The first two are of no importance provided that the patient soon seeks relief, but the last, owing often to the want of pain, is allowed to lapse, and various treatments are given, with the result that only harm ensues, much valuable time being lost, during which the growth proceeds and may get beyond the limits of removal. I have dwelt on this point because it seems necessary to emphasise the fact that it is wrong to make a diagnosis without having previously made every possible examination. For some reason or other

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this is frequently done in the case of rectal disease. It can only do harm to everybody concerned when the diagnosis is made in this perfunctory manner without careful investigation.

The diagnosis of hæmorrhoids is a simple matter, and one which the patient is often capable of making himself, pain, protrusion, and hæmorrhage being the three main symptoms. On examination possibly nothing is seen externally, but when there is a combination of external and internal hæmorrhoids the circumference of the anal margin has often a full and distended appearance, and the mucous membrane between the two sphincters may be slightly prolapsed and œdematous. Further, in cases of long standing, the hæmorrhoids themselves may be in a constant state of prolapse. Under these circumstances there is no difficulty in the diagnosis; but when nothing is of any diagnostic aid outside, it may be, and in fact often is, a matter of difficulty to the inexperienced to detect hæmorrhoids by digital examination, and it is also difficult for me to give any definite method by means of which any one can detect internal hæmorrhoids by digital examination, except by an experience of having examined a certain number of cases, in which case the condition of the normal mucous membrane will be known, and anything deviating from this will be recognised. The great point in these examinations is to remember that, with quite a few exceptions, all rectal troubles are situated either between the sphincters or at the level of the internal one, therefore the common mistake of trying how high the finger will reach should be avoided. It is by this method that so many things are missed in examining the rectum. When examining for hæmorrhoids the digital pressure must be quite slight in character, otherwise the injected mucous membrane which creates the hæmorrhoid will, by mere force of digital pressure, disappear, and nothing will be felt. A third method whereby diagnosis can be aided is in getting the patient to endeavour to force down any hæmorrhoids, which can then readily be recognised. These are the main simple methods by which the diagnosis can be made.

Occasionally hæmorrhoids are complicated with fissure, ulcer, or fistula. These complications are readily recognised by the methods which I have already described. Another feature in the symptoms of hæmorrhoids which is of considerable interest to both physician and surgeon is the anæmia, sometimes of a profound character, which exists in these conditions, and is solely due to the daily losses of blood, which, going on in some cases for years, renders the patient's condition a most distressing one, and complete invalidism is the eventual result.

Treatment.—The treatment of hæmorrhoids may be palliative or radical. To deal with the palliative treatment first, which often is most effective. Here the question of habit largely comes in. It is commonly recognised that everybody overeats and overdrinks, but particularly overeats. In middle life a large number of patients with hæmorrhoids exaggerate the condition, and practically create the symptoms by this habit of overeating, and conversely, if sufficient strength of mind can be brought to bear upon this habit, great benefit occurs to the patient, both with regard to his local condition and to his health generally. I cannot do better to illustrate this than quote the following case:—I was asked to see a patient of some fifty years of age who suffered very much from hæmorrhoids. He was a member of a City body, and as such thoroughly did his duty. This class of patient is not the one to choose for an operation, and we therefore persuaded him to keep in bed for some weeks, become a teetotaler, and to go on a rigid diet. This was done, with the result that every symptom disappeared, and any one seeing this patient to-day would hardly recognise him as the same man. He is in every way improved.

But there are a large number of much younger patients who suffer severely from hæmorrhoids and have not yet arrived at that condition of life when overfeeding has any marked effect upon the condition. At all events, it certainly does not produce it. In these cases it is as well for a few weeks to try some form of palliative treatment associated with diet. Ointments of various sorts are of use, particularly subchloride of mercury ointment of the strength of ten grains to the ounce. Subgallate of bismuth and hazeline ointment are at times of some effect. There is one ointment more commonly used than any other which I wish absolutely to condemn, and that is the ointment known as Unguentum Gallæ cum Opio. This does all the harm possible, and rarely any good. For medical treatment laxatives, when

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necessary, will, of course, be given, and for hæmorrhage doses of 10 minims of tincture of hamamelis have often quite a good effect. With one of these simple forms of palliative treatment the cases under discussion may be temporarily relieved, and sometimes cured. Another form of palliative treatment which in certain cases is most effective is the injection of the hæmorrhoids with a strong solution of carbolic acid. This form of treatment has been much written against in this country, and especially in America, but after several years' experience of it I am prepared to contradict most of the statements made with regard to the objections which are raised against it; but the proper cases, and those only, must be thus treated. The proper cases for treatment of this kind are prolapsing hæmorrhoids which are returnable; I emphasise "are returnable" because, unless this can be done, certainly disaster will follow this otherwise most effective treatment.

Given, then, a patient with hæmorrhoids which are prolapsed and returnable, the following method may, with all security, be followed out if for any reason a more radical operation is not desirable. A hypodermic needle is filled with a 10 per cent. solution of carbolic acid in glycerine. The needle is thrust into the hæmorrhoid to the depth of half an inch, and from 4 to 8 minims are injected into each hæmorrhoid, according to its size. There may be some bleeding from the needle puncture, but this is of no significance. The hæmorrhoids are then returned, and a pad and T bandage are fixed to prevent them prolapsing again. The patient should lie up, if possible, for two or three days, and the bowels should be confined for that period. The ultimate result of this injection is a considerable diminution of the hæmorrhoid, and, although it is in no sense a radical cure, yet months, and in some cases years, of relief are obtained.

When these prolapsed piles become strangulated they have a very typical appearance. The first thing to do is to reduce them, and under no circumstances whatever at this time should anything beyond this be undertaken.

After the reduction has been accomplished the patient should be kept in bed for a week, and care should be taken that the bowels are kept relaxed on and after the third day. If on examination at the end of this period the hæmorrhoids can still be felt it is better that they should be removed; but in various instances in my own practice the strangulation has in the event proved a cure, and no further interference has been necessary.

With regard to the operations for hæmorrhoids, I shall not deal with all of them, but propose only to discuss three, namely, (1) the clamp and cautery; (2) the ligature; and (3) complete excision, known as Whitehead's operation.

With the first of these my experience has been limited. The results obtained seem effective, but the possibilities of harm occurring are too great to my mind to make the operation desirable. So much tissue beyond that of the hæmorrhoid is caught up in the clamp and severely crushed; this is almost certain to slough, and, if there are three or four hæmorrhoids to be clamped, the eventual contraction may produce an absolute stenosis. Again, if the patient gets into a septic condition locally the results will be most disastrous. I have no doubt that most of us know of a large number of patients who have been operated on by the clamp and cautery, and have made an excellent and uninterrupted recovery, but in an operation of this kind one's business is to endeavour to avoid those risks which I have mentioned, and which, although only occurring in a small percentage, bring discredit to surgery.

The ligature operation is one which has been performed many hundreds of times at St. Mark's Hospital without any fatality; the ultimate results are extremely good, and when properly carried out with thorough antiseptic precautions, operation by ligature can be undertaken by any competent medical man. The operation is performed by first thoroughly dilating the sphincters. This does away with a certain amount of the after-pain, and brings the field of operation well into view. The various hæmorrhoids are seized with clamp forceps, and if the patient is on the side it is as well to operate on the lower side first. If this is not done the hæmorrhage from above obstructs the view for ligaturing below. A pair of cutting forceps is taken in one hand while the clip holding the hæmorrhoid is in the other, and the cut is made parallel to the bowel, care being taken to only include the mucous membrane, and with it, of course, the vessels. If a small portion of the skin is included in the cut, which should then lay open the superficial veins of the inferior hæmorrhoidal

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plexus, it will be found that the œdema which is so constantly occurring after these operations will either be entirely absent, or else minimised to a great extent. The end of the cut should be narrower than the beginning of it, so that the smallest amount of mucous membrane beyond the hæmorrhoid only is taken up when the ligature is applied. A good stout ligature of aseptic silk is now tied tightly close to the end of the cut. This must be carefully carried out. The hæmorrhoid is then removed, leaving sufficient pedicle to prevent the ligature from slipping. A certain amount of hæmorrhage will occur from the cut surface, but it is easily checked. Should, however, the vessels going to the hæmorrhoid be ligatured ineffectively or cut above where the ligature is applied, severe hæmorrhage will continue until this has been dealt with. A suppository of morphia is placed in the rectum, and a tube inserted surrounded by gauze. A pad of wool and a **T** bandage completes the dressing. The after-pain of this operation is sometimes most severe, and may require various hypodermics of morphia for the first twenty-four hours to allay it. I believe it is the practice of some surgeons not to give any opiate in these cases, but I cannot see any sufficiently good reason for doing this. The pain lasts for twenty-four hours; the discomfort of the patient after this is not very great, and the only remaining bad time is the fourth morning after the operation, when the bowels act. This is effected by a quite strong purgative given by the mouth, and I think there is nothing better than castor oil, associated with this is an oil enema of about sixteen ounces, to which are added two ounces of castor oil. This should be given slowly with a long tube, and should be previously warmed. From now on the patient is quite comfortable, and the only essential is that the operation area shall be kept scrupulously clean. The other point of importance is the horizontal position, which should be kept to in most cases for three weeks. The ligatures come away at about the eighth day, after which there is a granulating surface which gradually heals and contracts up, assisted by some of the well-known applications, such as subchloride of mercury ointment, hazeline ointment, zinc, vaseline, and the like.

The sources of danger in this operation are to be well borne in mind. They are hæmorrhage, sepsis, contraction, and recurrence. The latter is not in itself a danger, but is an undesirable possibility. Hæmorrhage from the rectum is one of the worst forms of secondary hæmorrhage that occurs, because unless, sometimes even when, proper precautions have been taken, the hæmorrhage continues steadily until quite an alarming amount of blood has been lost, and suddenly the patient shows signs of this and may rapidly die. Fortunately the hæmorrhage is usually discovered before such a dire result occurs, but under any circumstances it is an extremely serious disaster, and one which requires all the resource one can bring to bear on it. The worst cases are those in which some small vessel has been overlooked, and the plugging has not been applied so as to compress it. At the time it may have ceased to bleed, and the post-operative condition of partial collapse will prevent any serious loss for some hours; but as reaction takes place and the tension increases the vessel bleeds freely, and the patient suddenly becomes faint—is observed to be pale, sweating, the pulse is fleet and fluttering, and the patient keeps on yawning. This particular symptom may be the first to which attention is called, and should always be regarded with suspicion. The only thing to do under these circumstances is to give the patient an anæsthetic if possible, and then to dilate the sphincter, when it will be found that a very large quantity of blood which has slowly been accumulating in the rectum will escape. A hot saline douche should be injected, and careful investigation made for any bleeding vessel, which, if found, must be secured. If this is not found the only thing to be done is to carefully pack all round. A tube is to be passed in four or five inches. Dependent on the amount of blood lost it may be desirable to transfuse with saline, and if an anæsthetic has been given subcutaneous transfusion in the axillæ should certainly be done.

The *signs of sepsis* are pain continuing after the usual time, the temperature rising when it should fall, and a locally inflamed condition beyond that which is due to the operation. There is also that peculiar distressed condition and restlessness which are two features in all cases of sepsis most suggestive, and to my thinking, not sufficiently recognised. Another point is the condition of the skin generally. In these cases the skin is dry and harsh, quite a different condition from the skin of the patient who, in spite of a temperature, has no

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sepsis. In treating this condition no time should be lost, and the treatment must be carefully carried out by a conscientious and good nurse, for if it is not recognised and is allowed to become chronic a most lamentable condition of affairs supervenes. The treatment consists of hot boracic baths, hot boracic fomentations, and having the affected area frequently washed with injections of solutions of perchloride of mercury, or some such strong germicide. Further, the two sides of the bowel should be kept apart by an antiseptic dressing. The good effect of the treatment is soon apparent in the altered condition of the patient, the dropping of the temperature, the absence of pain, and a more or less profuse discharge of what used to be termed healthy pus. I use the term here because it serves to distinguish this discharge from that which occurs when the septic condition is chronic. Under these circumstances the discharge is sanious in character and of thin consistency.

When allowed to become chronic this ulceration proceeds for weeks and months and years, making the patient's life one of great misery, associated as it is with much pain and discomfort. Finally it often leads to a bad form of fibrous stricture. Of this I shall have more to say later on.

Contraction occurs occasionally when, owing to the number of hæmorrhoids which have been tied, the area of granulating tissue has almost, possibly entirely, encircled the bowel, and this may lead to a permanent stricture unless it is recognised in the early stage; therefore it is most essential that a digital examination should be made about the third week, and, if necessary, a short conical rectal bougie must be inserted a few minutes at a time for some days at night-time.

Recurrence is not an unusual thing after an operation by ligature. I do not say that if the operation is thoroughly carried out such a thing is possible; but in my own experience, and in that of others to whom I have spoken, it is not uncommon for such recurrences to happen. This brings me to the third operation, namely, the complete excision, or Whitehead's operation.

The advantages claimed for this operation are: (1) the extirpation of that part of the mucous membrane which may be termed the pile-bearing area, and, therefore, the impossibility of a recurrence; (2) the impossibility of rectal hæmorrhage occurring, inasmuch as the whole field of the operation is for after-treatment in full view of the operator; (3) the period of convalescence can, if necessary, be materially shortened; (4) the pain after the operation, although not insignificant, is usually less than after ligature; (5) the possibility of sepsis is by no means so great, and should it occur it is much easier dealt with after this operation than after the ligature.

In cases of marked anæmia due to hæmorrhoids I consider this operation the only one which should be done, because the blood in these cases is so altered that it refuses to clot, and constant oozing is likely to occur. Again, owing to the general feebleness, the tissues are apt to slough, and secondary hæmorrhage and sepsis are more likely to occur. The benefit of the operation in these cases is well illustrated in the two following instances.

Miss A—, a patient sent to me by Dr. James Galloway, had suffered for many years with profound anæmia, and had been treated during that period by various medical men with all the various remedies which are used in these cases. Dr. Galloway having recognised the fact that the patient for a long time had almost daily suffered from small losses of blood from the rectum, found on examination that this was caused by hæmorrhoids of a somewhat extensive character. The patient, when seen by myself, had a typical blanched, saffron-coloured skin, which almost suggested the condition of advanced malignant disease. The pulse was consistently feeble both in volume and character, there was a well-marked anæmic bruit, and all the other symptoms of anæmia. The patient's condition was so bad that it was evident any further loss of blood was to be checked as soon as possible. What was the best to do under these circumstances? I was myself in favour of Whitehead's operation for the reasons that I have already given. The one point against it was the possibility of excessive hæmorrhage occurring during the operation. However, of the two risks I concluded that with proper precautions this was the lesser, and with the precaution of having all the materials ready for saline infusion if the necessity arose, I proceeded to operate. It was at once noticed when the mucous membrane was incised that the blood was very watery in consistency and did not readily clot. Only a

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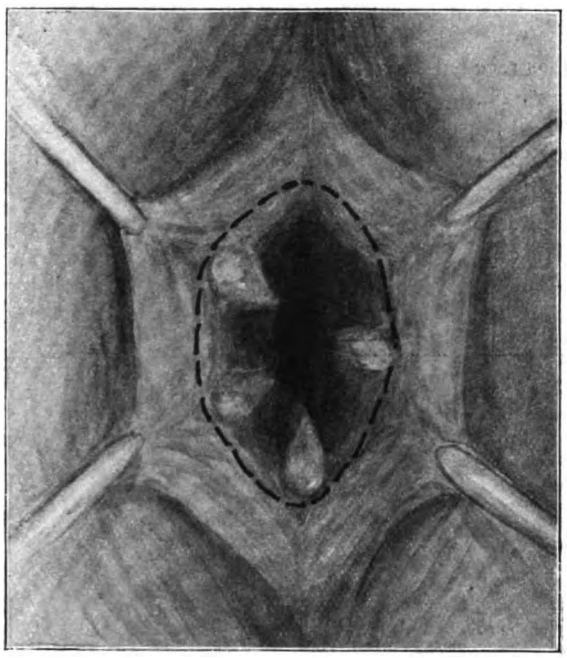
small quantity of blood was lost at the operation, and the patient made an excellent recovery. All signs of anæmia have now disappeared, and the patient is a perfectly healthy woman.

The other case was sent me by Dr. Owen Lankester. This was a married lady of nearly

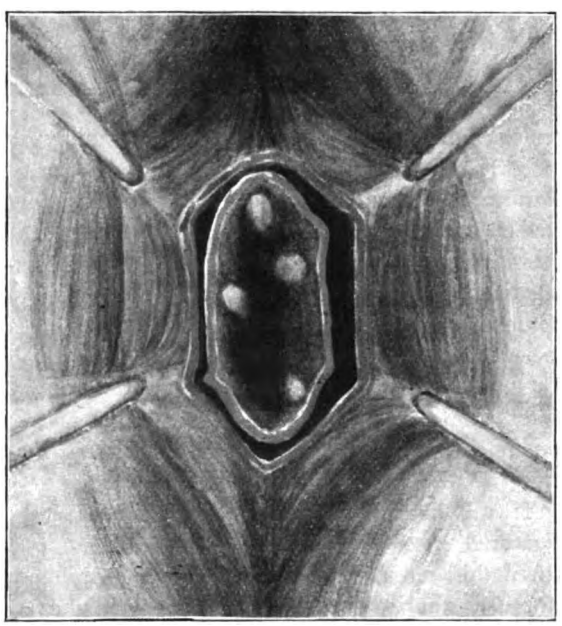
FIG. 1.



fifty years of age whose condition was somewhat similar to the other, but not so marked in degree. The same operation was performed in this case, and the result has been equally gratifying.

As to the method of operating it should be said at once that this operation, contrary to the last of ligature, is one which demands intimate knowledge and constant practice of surgery, and should not be undertaken under any circumstances except by an experienced surgeon. The results which have come under my notice when this axiom has not been complied with have been disastrous. The whole of the external sphincter and a large portion of the internal have been removed, and certainly the operation was sufficiently radical, but the patient's condition was deplorable, a marked prolapse and permanent rectal incontinence being the result. The length of time taken for the operation is often unduly long. Experience alone will prevent this, but at no time should the operation take longer than twenty-five minutes, from fifteen to twenty minutes being the average. The operation is performed with the patient in the lithotomy position. The sphincters are well stretched and the hæmorrhoids brought into view. Clip forceps are placed upon the skin just external to the mucocutaneous margin above, below, and either side. This margin is next incised all round, the parts being kept fairly tense by the clip forceps on the skin, and the operator holding the mucous membrane with dissecting forceps (*vide* Figs. 1 and 2). A scalpel separates the mucous membrane down to the external sphincter, which is recognised by the circular muscular fibres. The knife is then laid aside, and a pair of curved scissors taken. The separated mucous membrane is drawn forward (*vide* Fig. 3) by the operator's finger and thumb, and the adherent muscles, both internal and external, are separated by the scissors until the whole of the mucous membrane has been freed, and can be easily pulled down to the cut margin, having the

FIG. 2.



area containing the hæmorrhoids well outside. During this part of the operation I have recently adopted the plan of having a small stream of warm water and hazeline running over the wound. This does away with any necessity of sponging, any vessel of consequence which may be cut is

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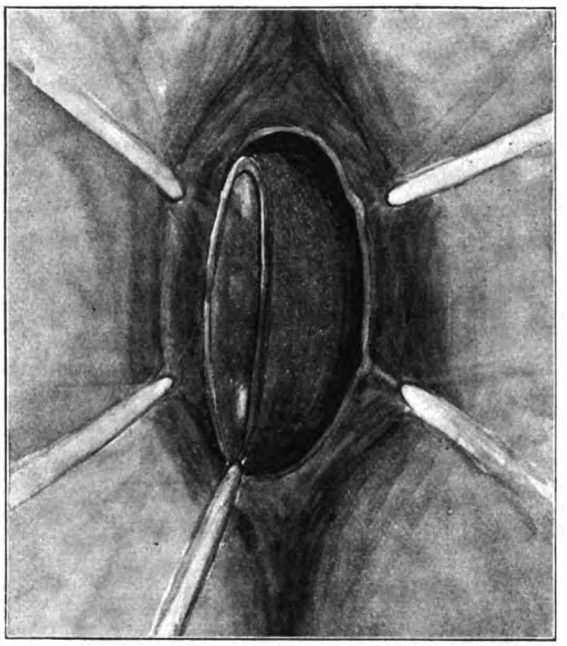
immediately seized, and the hæmostatic effect of the hazeline acts well on the cut surface. By this means considerable time is saved. The next step in the operation is the removal of the separated

FIG. 3.

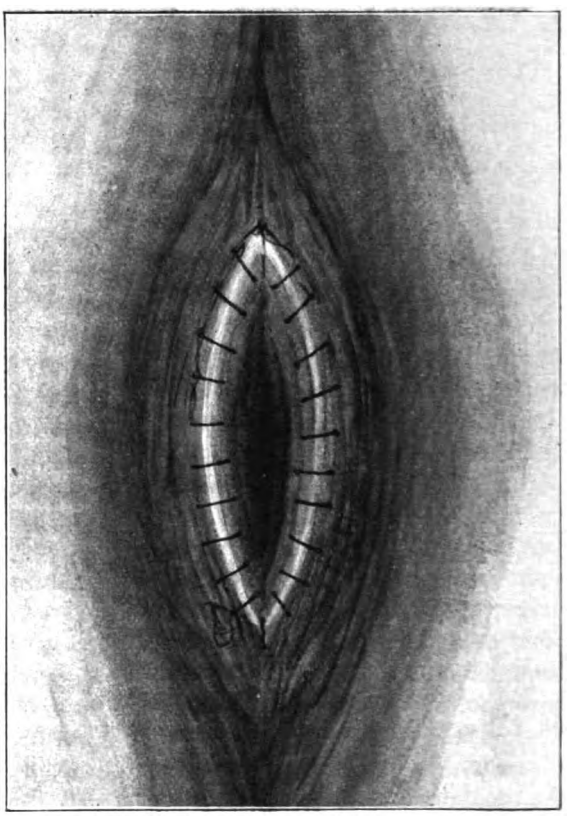


mucous membrane. This is done as rapidly as possible with scissors and competent assistants' hand clips, which are immediately applied to any vessel. It is during this part of the operation that hæmorrhage may occur unless these clips are immediately applied. When the whole circumference of the separated mucous membrane has been removed, the cut mucous surface is sewn to the cut margin of the skin by one continuous interlocking suture of catgut which completely encircles the bowel, and this terminates the operation (*vide* Fig. 4).

It will be noticed that no vessel has been ligatured, and should any hæmorrhage still continue after this circular stitch has been completed, another suture is passed by means of a curved needle somewhat deeper, and includes the sphincter when it is tied up. One or two of these effectively checks any hæmorrhage, but, as a rule, they are not necessary, and should not be used unless the necessity is fairly obvious because the pain is much increased by deeply placed ligatures.

The after-treatment in these cases is quite simple. The wound is kept as dry as possible, dry powder, either iodoform, calomel, or zinc oxide, and boric acid, is applied, and some absorbent dressing with a pad of cotton wool is all that is necessary. This, however, must be as often renewed as any marked oozing occurs. An aperient is given on the evening of the fifth day. accompanied the next morning by an oil enema. When this has operated and the wound is examined it will be noticed that the mucous membrane has receded from the skin margin for about half an inch, more or less. This invariably occurs, and need not cause any alarm. The exposed interval is dressed with dry dressings and powder as before, and gradually granulates up. The patients are better kept in bed for a full fortnight, if not longer. In one or two instances where, owing to some

FIG. 4.



business emergency, it has been imperative for the patient to get up after the first week I have noticed that a certain degree of contraction takes place. This contraction is the one drawback to

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the operation, if such it can be called. In the majority of cases it is so slight that no attention is paid to it; but even in those cases where it causes some slight temporary stenosis this is easily overcome by the passing of a vulcanite conical rectal plug and keeping it in place for a few minutes at night-time. If this is done for a few nights the whole of the contraction will disappear, and does not recur. I have never had a case in which such a contraction has caused anything resembling permanent stenosis.

It will be thus seen that the operation has a great deal to recommend it in suitable cases, and it is a radical measure. This cannot be said always for the ligature, as in many cases the operation has to be repeated. Let me conclude by saying again that Whitehead's operation is an extremely good one, and a good piece of surgery when properly carried out, but may prove disastrous when undertaken by the inexperienced.

Prolapse and Procidentia.

By this is understood a protrusion of the mucous membrane which, although in most cases reducible, yet soon returns again to the prolapsed condition. The difference between prolapsus and procidentia is a more or less arbitrary one, but the usual meaning of prolapsus is when the mucous membrane alone descends. When, in addition, the whole thickness of the intestine protrudes it is called a procidentia. Prolapsus is a common trouble in small children, especially of the poorer class, and is often seen in our every-day hospital practice.

Causes in Children.—Among the poor the commonest cause is bad or improper food; this, by its irritating action on the intestine, produces diarrhœa accompanied with great straining, and a continuance of this causes the prolapse. Other causes are polypus, ascarides, phimosis, and vesical calculi, and it will be noted that all these conditions are conducive to straining. The bad custom of many nurses to produce a daily action of the bowels is perhaps as common a cause of prolapse as any, and when prolapse is due to the last-named cause the sphincters are more relaxed and flaccid than usual. The reason for this being that the condition has probably existed some time before its discovery.

Causes in Adults.—This condition is not a common one in adults, and is oftener met with in women than men. The common causes are hæmorrhoids, polypi, enlarged prostate, calculus, parturition, and, in fact, anything which causes great straining or weakening of the muscles in this region. The aspect of the prolapsus depends upon whether it is of a chronic or sudden condition. In the latter the prolapsed mucous membrane becomes gripped by the contracting muscle, soon becomes livid in colour, and may become gangrenous. A child under my care in the hospital had this condition, and the whole ring of the protruding intestine sloughed off. When the cause is a chronic one the mucous membrane is normal in colour, as in these cases there is no gripping of the sphincter. There may be superficial abrasions, but nothing beyond this.

Treatment.—In children this depends upon the cause. If it is a recent prolapse and a sudden one which is caught and held by the contracted sphincter the prolapse must be reduced. This is readily done by placing the child prone on the nurse's lap and applying firm, gentle pressure for a minute or so when, with a little manipulation, the reduction is easily brought about. When the prolapse is chronic the child is kept in bed, and the bowels should only act when the child is lying down. The mucous membrane should be sponged with cold water and have some astringent lotion, such as alum, ten grains to the ounce, applied. The bowel should be returned, and a pad and perineal bandage applied. In bad cases the child should be kept in bed and be encouraged to lie on its face. Constitutional treatment should be carefully carried out. Diet is a matter of importance, and must be well considered. Judicious doses of Hydr. c. Cret. and rhubarb may be given; cod-liver oil is an excellent thing in these cases, both for its nutritive properties and because it helps materially to prevent constipation. Under such treatment as this most children get well, although occasionally the recovery is slow. But if after a fair trial prolapsus still occurs, some more energetic form of treatment is necessary. The free application of nitric acid has been advocated, but personally I should be loth to recommend this as an application for an otherwise healthy mucous membrane. The actual cautery is much more effective, does relatively very little damage to the mucous membrane, and is not, when properly

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carried out, followed by any suggestion of stricture. As a last remedy, when everything else has failed, the prolapsed mucous membrane must be removed by a method similar to that already described in the complete excision of hæmorrhoids, but great care must be taken in children, greater even than in adults, to avoid damaging the sphincters, which in these small patients are, comparatively speaking, slight, and not so easily recognised.

Procidentia recti, which usually occurs in adult life, may be partial, complete, or intussuscepted. The treatment depends to a great extent upon the variety. Other causes than those already mentioned for prolapsus are weakness of the pelvic supporting muscles, namely, the levatores ani, and associated commonly with this is a long meso-rectum. The treatment of this condition may be palliative or radical. The former is not of much benefit, excepting in quite slight cases. Ball and Cooper speak well of a conical vulcanite pessary with a narrow neck. This method is credited with having effected a complete cure. Another method of treatment which is written about, but not, as far as one can make out, with any strong desire of advocacy, is the submucous injection of various irritating fluids, the aim being to procure inflammatory matting of the various coats of the bowel, and by this means to prevent the procidentia. I do not regard this as good surgery, and will not go further into it.

The operations which have been undertaken from time to time are fairly numerous, and all more or less ingenious. Verneuil raises the bowel and attaches it to the sacrum. Macleod, of Calcutta, and Allingham recommend an incision in the abdomen, and pulling up and attaching the sigmoid to the abdominal parietes. Treves and Mikulicz have both devised operations which in principle are the same, the object being to remove the whole of the prolapsed intestine, bringing the cut edge of the mucous membrane down to the skin margin in a similar manner to Whitehead's operation. This method I have adopted in various instances in my own practice, and have every reason to be satisfied with it.

The actual cautery is in a large percentage of cases undoubtedly the most effective and least dangerous method which can be adopted in all forms of prolapse, and should generally be attempted before any more serious proceeding is undertaken. The intestine being pulled down to its fullest extent a full-sized Paquelin's cautery at a dull red heat makes four longitudinal stripes, deepening as they come near the base. These stripes are made anteriorly, posteriorly, and on both sides. The burning process can be quite thoroughly carried out, and what appears as a very great destruction of tissue at the moment turns out to be, comparatively speaking, a slight matter. The result of this treatment is most satisfactory, and if a certain amount of return occurs, which is to be expected in bad cases, another application and even a third may be made with increasing benefit to the patient.

The after-treatment consists of rest in bed for some weeks, during which the horizontal position is maintained under all circumstances.

Ulceration.

In a paper which was published in the 'British Medical Journal' in October, 1900, on "The Causes and Treatment of Non-malignant Stricture of the Rectum," I dealt somewhat largely with this question of rectal ulceration, and I will here merely refer to the main points with regard to this condition.

The causes of ulceration are commonly said to be syphilis, tubercle, and dysentery, and of these the first has the greatest reputation. Obstetric trauma and operations are mentioned vaguely as occasionally causing ulceration.

With regard to the frequency of syphilis as a cause of ulceration I can only repeat what I have already written, namely, that these conditions of ulceration are much more common in women, that the patients show no other signs of syphilis, and the most careful questioning fails to obtain any evidence of disease.

With regard to dysentery there is no evidence which I have been able to obtain that will show that dysenteric ulceration commonly affects the rectum.

Tuberculous ulceration occurs, but presents aspects of its own, and is usually associated with fistula, and with this we have already dealt.

The most common form of ulceration which has come under my notice during the years that I have been in St. Mark's has been that due to sepsis. By this I mean ulceration due to some septic discharge from the outside attacking the greater

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portion of the mucous membrane and so progressing, or, what was a common cause in the past, post-operative sepsis. Another cause of some of the worst cases of ulceration is a protracted labour, when the child's head becomes fixed in the pelvis for some hours, and by direct pressure causes ultimate destruction and ulceration of the mucous membrane. This separates, and the denuded surface, exposed as it is to the continual contact of fæcal matter, becomes septic, and progressive ulceration starts. This is a common case which comes under one's attention at St. Mark's. Until lately I had not seen a single instance of ulceration of the rectum which could in any way be attributed to any other cause but sepsis, and this one isolated case I am as yet unable to classify.

The main symptoms of this septic ulceration are quite typical, and when once pointed out can in future be easily recognised. Around the external sphincter the skin and muco-cutaneous margin are swollen and œdematous, and often between the two œdematous portions the skin will be found fissured and ulcerated. On separating the parts the mucous membrane may appear for a small area without any sign of ulceration. On examining, a proceeding which always causes great pain, it will be found that ulceration, commencing just within the bowel and spreading often around the whole surface, has invaded the mucous membrane to a varying extent; and dependent on the length of time during which this has been in progress the extent will be greater or less. The discharge is typical—blood-stained, thin, and often offensive. Defæcation is a most painful process, and on this account generally incomplete.

When the ulcerative process has ceased to extend chronic thickening occurs at the upper limit, which eventually becomes a fibrous stricture, the calibre of which depends upon the amount of fibrous thickening and contraction. Associated with this condition there have been various cases under my care in which joints have become filled, hot, and tender, accompanied with a general rise of temperature. These symptoms may occur in two or three joints, but the knee is most often affected. If the skin margin is ulcerated the glands in the groin are usually enlarged. The indications that a recent operation has become septic are a rise of temperature which is continuous, extreme pain at the wound of a throbbing character, some swelling of the surrounding parts, and a blood-stained discharge. In severe cases there is a rigor. This condition, if not immediately and rigorously dealt with, soon assumes the chronic condition of which the symptoms have just been enumerated. Many patients who have suffered from this septic ulceration have on account of the symptoms which they complain of been treated for dysentery. This mistake can easily be avoided by local examination and inspection. The importance of recognising the disease as early as possible cannot be over-rated. The quicker it is recognised the less progress will the ulceration have made, the less tissue will therefore be involved, and the repair when it occurs will not produce any marked condition of stricture, such as invariably occurs in advanced cases.

Treatment.—When a patient after operation exhibits those symptoms already given indicative of a septic condition fomentations should be applied externally, and the wound thoroughly and frequently irrigated with perchloride or biniodide of mercury solution, or a solution of peroxide of hydrogen mixed with an equal quantity of warm water may be used. The horizontal position must be strictly maintained, fluid diet is desirable, and it is as well to give some intestinal antiseptic, such as salol. Scrupulous cleanliness must be observed, and the frequent irrigations should on no account be omitted. The indication that the acute condition is over is shown by the temperature coming to normal, disappearance of the pain, and the change in the nature of the discharge, which becomes purulent and not blood-stained. On examination now it will be found that healthy granulation tissue is present, and all that is necessary is that this shall be kept clean. Delay in recovery in these cases is not particularly prolonged, but on no account should any hurry be exhibited in allowing these patients to assume the perpendicular position. When the condition is a chronic one the treatment is much more difficult, longer, and often complicated. The difficulty in these chronic and advanced cases is to get the ulcerated area into a healthy healing condition. This is mainly due to the fact that the contents of the intestine are constantly re-infecting the affected area. It is possible, however, eventually by great perseverance to obtain this result in a certain number of cases, but a proportion will not, even after months of treatment, alter their character

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in the slightest. It has therefore become my custom if after two or three weeks local treatment no improvement shows itself, to perform a temporary colotomy. By this means the contents of the intestine are diverted from the affected area, and this can be thoroughly treated when the ulceration has finished and the healing fibrous contraction has been assumed; the question of further treatment is a matter which must be dealt with according to the extent of the contraction. Should contraction be of such narrow dimensions that it will be necessary always to pass a bougie to keep the parts sufficiently dilated, and this is the rule, the best treatment is to excise the whole of the mucous membrane from the muco-cutaneous margin up to and beyond the fibrous stricture, which, as I have already said, usually indicates the limit of the ulceration. When the dissection of the fibrous stricture from the surrounding parts has been effected, and healthy mucous membrane arrived at, this latter should be further separated until it can be brought down to the margin of the external sphincter, to which it should be sutured.

I have performed this operation now five times. On the first occasion without a preliminary colotomy; the others were preceded by a colotomy, which was subsequently closed. The limit to which the amount of bowel can be removed is a debatable point. Five inches is as far as I have been able to go, and in three cases the peritoneum has been freely opened. This in a way greatly facilitates the operation when once the strictured portion has been overcome. The operation is a difficult and tedious one, often associated with great loss of blood, so that at the end of an operation which may take the best part of two hours the patient is found to be much collapsed, and great care is required in the nursing of these patients for the forty-eight hours following the operation. Saline injections should be administered before the patient leaves the operation table, and strychnine should be given hypodermically every two hours until the condition of collapse has been overcome. In a certain number of these cases the limit of the ulcerated area may be so high that it is impossible to know how far it extends. Under these conditions a preliminary colotomy is a matter of necessity because of the possibility of being unable to entirely remove the whole of the strictured portion. In two instances in which I had to do this the patients, having regard to the condition of misery in which they lived previous to the colotomy as compared with their present state of health, refused on any account to have the colotomy wound closed.

The excision of this ulcerated area is undertaken from ten days to a fortnight after the colotomy, provided that in that time the ulcer has shown signs of repair. Another fortnight must ensue before the colotomy wound is closed. The method by which this is done has already been detailed in the paper which I read at Ipswich, but briefly the points are—(1) Separation of the junction of the mucous membrane with the skin. (2) Dissection of the parietal peritoneum to which the peritoneum of the colon has become adherent. This is done freely, so that the colon is quite loose with no tension; and it will be noted that the general peritoneal cavity has not been opened. (3) The opening into the bowel is closed by a continuous *transverse* suture of the mucous membrane. An interrupted series of Lembert sutures is next put in rather less than a quarter of an inch from the continuous suture and extending well beyond each end of the continuous suture. (4) The abdominal parietes are brought together by interrupted fishing-gut sutures which do not include the skin. This is a matter of some importance. If the skin edges are approximated and unite any effusion underneath this, which can hardly be expected to be otherwise than septic, will be unable to escape, and an abscess is the probable result. When the skin edges are not brought together the fluid escapes and is absorbed by the antiseptic dressing, the wound heals rapidly, and the mere fact of the skin not being opposed makes no practical difference to the ultimate scar.

There is nothing particular to note in the after-treatment beyond that no attempt should be made to confine the bowels; in fact, it is my custom to give oil on the third night after the operation to prevent anything like constipation. This formidable treatment may sound excessive, but any one who has seen a case of bad ulceration of the rectum will, I think, agree that anything which can be done to alleviate the life of misery in which these people exist is not only justifiable but called for.

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A CLINICAL LECTURE

ON

"SEBORRHŒA."

Delivered at St. Bartholomew's Hospital, June 30th, 1902.

By J. A. ORMEROD, M.D., F.R.C.P.,

Physician in Charge of the Skin Department of the Hospital.

GENTLEMEN,—At the beginning of last week, and until the bad news fell upon us which altered the whole course of that week, I had made up my mind that it would be useless to ask patients to come here to-day, for I hardly supposed they would have finished celebrating the Coronation. So I felt obliged to give you something less in the shape of a demonstration and more in the shape of a lecture than I have hitherto done, and as I have not had time to alter the arrangement, I must ask your pardon for giving you a set lecture rather than an object-lesson.

The terms seborrhœa, seborrhœic eczema, seborrhœic dermatitis, are in very common use, because the condition which they are intended to denote is very common. It is now almost impossible to avoid using some of them, yet I cannot help feeling that their connotation is somewhat uncertain, and it is difficult without reference to the opinions of those who originated them to see why they should be used. I propose to-day to consider what they mean.

It is not always unprofitable to consider the meaning of a name, for even in science this may become so rapidly modified that the facts—it may be important facts—upon which it was based become forgotten. In every-day life, of course, this is constantly happening. For instance, I dare say the coming generation, or perhaps even some of you, would be unable to say why certain politicians or certain newspapers are called "jingoes," or why a certain method of treatment is called "boycotting."

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The term "seborrhœa," as you know, is used to denote two sets of clinical facts. When we classify it as "seborrhœa oleosa" we mean to describe an appearance which is seen in its most marked form on the scalp of children. Yellow greasy crusts form on the vertex without any apparent inflammation of the skin beneath, except perhaps such as may be due to a secondary irritation. They consist, it is thought, of secretion from the sebaceous glands produced in excess and more or less dried upon the skin surface. On the face too, particularly on the sides of the nose and the middle third of the face, the secretion may be seen, generally in a more fluid and oily form, though here too crusts may form; or there may be no visible secretion, but the skin is simply greasy and blotchy. The affection on the face is perhaps most frequent in people with coarse dark skins, who may be also the subjects of acne. The type, I should think, among adults is the middle-aged Jewess from Whitechapel or Spitalfields. Secondly, when we speak of seborrhœa sicca we mean an appearance also seen most frequently on the scalp, but not so much caked, yellow, greasy crusts as whiter, dryer, finer scales, either loose in the hair as ordinary scurf or dandriff, or in bad cases clinging in a thin uniform coating to the scalp. But the affection does not stop on the scalp. It may creep outwards as a brownish branny rash with a rounded or sinuous margin upon the forehead or occiput, and may invade in scattered patches the face, neck, shoulders, trunk, or indeed any part of the surface. Generally it maintains a descending course, but I do not think this is invariable; nor, indeed, is it absolutely necessary that the scalp should be the starting point, though no doubt it is generally so. The rash mostly takes the form of flat papules or patches, pale red, with a yellowish or brownish tint, covered with fine scales, and dry; but it may be modified by spreading into circinate or ringed forms, or may be varied by the part of the body (moist or dry) where it occurs, or by secondary changes of a frankly eczematous kind. It is liable to be confounded with early syphilids, tinea circinata, tinea versicolor, pityriasis rosea, or psoriasis.

Why are these two sets of symptoms classed together? And why have they both received the name seborrhœa? A recent authority, whom I shall presently mention, says indeed that they have nothing to do with each other. Nevertheless seborrhœa oleosa and seborrhœa sicca have been so associated, and apparently for the following reason:—The oily form of the disease was thought, and is still thought, to be caused by excessive secretion from the sebaceous glands; the sebum was considered to be the source whence the skin drew the fat and oil necessary for its due lubrication; and excess of sebum produced an undue greasiness of the skin, or even the visible oil and grease caked upon the surface. In this connection the name seborrhœa seems natural enough. It was next pointed out that as the sebum was produced by the actual breaking up of the cells of the sebaceous glands it must consist not only of free oil and fat, but also of scales and *débris* derived from the walls of the cells. Hence (it was said) we can have two kinds of seborrhœa according to variations in the quality, rather than the quantity, of the sebum—an oily form if fat predominate, a dry form if scales and epithelial *débris* predominate. This sounds a plausible reason for associating the two things under the name of seborrhœa; but it overlooks the fact (or at least what good authorities assure us is the fact) that the scales of seborrhœa sicca correspond in appearance to the epithelium of the general surface, and not to the remains of glandular cells.

But you will say, What about "seborrhœic eczema?" What does that expression mean, and how did it come to the front? I believe it came to the front through the agency of Unna, whose views on the subject we must now consider. I take them from his great work on the histo-pathology of the skin, but I must warn you that his phraseology and style are not always easy to understand.

First of all, his opinion is that the oil which naturally becomes incorporated with the skin, and which appears to be in excess in the condition called seborrhœa, is not secreted by the sebaceous glands but by the sweat glands. If that be so, the word is etymologically a mistake. But we may let that pass, partly because most authorities do not agree with Unna in this particular opinion, partly because he himself continues to use the word.

Secondly, when ought we to speak of a seborrhœic eczema in contra-distinction to seborrhœa? I confess that hitherto when any of you have asked me this question I have replied that

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when the original pale, dry, scaly patches become reddened, develop vesicles, weep, become raw or crusted, then an eczema has set in, superimposed upon and started by the seborrhœa, and therefore properly called a seborrhœic eczema. This view is perhaps simple and intelligible, but it is not, I gather, Unna's view, and that is not what he means by seborrhœic eczema, for he thinks that the whole process is eczematous from the beginning. What, then, does he lay down as the characteristics and criteria of an eczema? Certain histological features which, if you will be patient with me, I will try very briefly to explain.

There are three histological processes, he says, which go on in chronic eczema (acute eczema is for the moment not considered), and on the preponderance of one or other of these depend the clinical characters of the eczema. The first is called "para-keratosis." Para-keratosis means an interference with the normal process whereby the rounded or polygonal cells of the rete mucosum become transformed into the flat, horny cells upon the surface of the skin, a process sometimes called "keratosis," sometimes "cornification," sometimes in English "horning." Just as in rickets the normal transformation of cartilage into bone is stopped, so here there is interference with the normal transformation of the epithelial cells into those of the horny layer. The cause of this interference is, according to Unna, an œdema (parenchymatous swelling) of the cells which are about to be transformed; and the result is that instead of completely horny, dry, flat cells on the surface which fall off one by one imperceptibly, there are found incompletely dried cells which stick together in the form of scales. The result is a scaly eczema. It is a curious, and perhaps rather puzzling consideration that what appears to be clinically a dry form of eczema should be started by œdema of the epithelial cells.

The second process is proliferation of the cells of the rete mucosum, which by increasing the volume of the epithelial layer causes, *inter alia*, the appearance of papules and plaques on the surface. From this process (which he calls "acanthosis") there results clinically a papular eczema.

The third process is effusion of fluid into the lymphatic spaces between the epithelial cells, an interstitial œdema that is, as opposed to the œdema of the cells themselves. The lymphatic spaces become dilated, and this dilatation may go on to the formation of localised cavities, that is to say, vesicles. This process, which he calls "spongy transformation of the epithelium," is responsible for the weeping and for the vesicle formation which may be observed in chronic eczema.

Now a seborrhœic skin contains an undue amount of fat, and, according to Unna, the effect of this fatty condition will be to limit the production of the third of these factors, *i. e.* the process of interstitial œdema, when an eczema sets in, leaving the first two processes to go on as usual. A seborrhœic eczema is therefore only an eczema so modified by a previous fatty condition of the skin that there is no weeping or vesiculation, but only formation of papules and scales. And this is just what we see in so-called seborrhœa sicca.

Let us now turn to the views of another famous living dermatologist, I mean Sabourand. I take them from a report of his speech at the Cheltenham meeting of the British Medical Association last year. We expect the statements of a French savant to be clear, precise, and suggestive; and they are so in this instance.

There is, he says, only one type of disease which should be called seborrhœa, and that is the type which we distinguish as "seborrhœa oleosa." The greasy crusts and the oily secretion observed in this disease come, as is generally thought, from the sebaceous glands, not from the sweat glands. The "elementary lesion" of this, the true seborrhœa, is a little fatty plug, which can be easily squeezed from the gland, and which contains, he says, a colony of special bacilli. These bacilli he regards as the cause of the disease. He brings this seborrhœa into relation with several other skin diseases, as follows: When the little fatty plug just mentioned becomes encysted and shut up in a gland it constitutes a comedo, and this encysted form of seborrhœa is identical with ordinary acne. If the encysted plug becomes infected with cocci, the acne becomes pustular, or indurated, from deeply seated inflammation. If the cocci are very virulent, the follicle necroses and forms a furuncle. Further, he attributes ordinary (coronal) baldness to an upward spread of the seborrhœic bacillus into the hair sacs of the vertex. (Most English dermatologists do not agree with this view.) Lastly, in common with others, he thinks that seborrhœa

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produces a soil suitable for the development of sundry other skin diseases, such as warts in old people, or molluscum contagiosum in the young.

But with regard to seborrhœa sicca he makes the following distinct propositions:—(1) That it is not a seborrhœa at all, and has nothing to do with seborrhœa, nor even with a fatty condition of the skin, except so far as the two things may be coincident accidentally. (2) That consequently the name seborrhœa ought to be dropped, and may be replaced by the old name pityriasis, which, as you know, means bran-like scales, or scurf. (3) That these fine scales, or scurf, which are the clinical characteristic of the disease, consist of exfoliated epidermis from the surface, and not of *débris* from the cells of sebaceous glands. (4) That the disease is due to a micro-organism, probably either a coccus or a "bottle bacillus," but not to the special bacillus which he says causes seborrhœa oleosa.

Most authors I notice agree to the statement that seborrhœa sicca is infective, that is, has a microbic origin; though I suspect more on account of the general course and spread of the disease than from any definite bacteriological proof. Indeed, I should think that proof of the bacteriological origin of many skin diseases was made difficult by the very abundance of the material In this disease, I read in Malcolm Morris's book, one bacteriologist has isolated no less than eighty varieties of microbes. Well, there may be safety in numbers, and our skins appear simply to swarm with these things.

I have talked so much about names that you will think I am far gone in the dermatologist's disease, that is to say, a love of nomenclature for its own sake. Not so, for as long as you recognise the clinical appearances of so-called seborrhœa and know how to treat it I do not much care what you call it. You may shelter yourself behind the great authority of Unna and call it a seborrhœic eczema, or, if you think the seborrhœic theory exploded, you may call it a "dry eczema;" or a "seborrhœic dermatitis;" or a "desquamative catarrh;" or you may call it by the good old name of "pityriasis," provided you do not thereby confuse it with other diseases, such as pityriasis rubra, pityriasis versicolor, etc., with which it has nothing to do.

All that I ask is, that whatever name you employ you should have some idea of what that name means, and it is in the hope of helping you towards this that I have troubled you with this lecture.

WITH MR. JACKSON CLARKE

AT THE

MEDICAL GRADUATES' COLLEGE AND POLYCLINIC.

MARCH 26th, 1902.

GENTLEMEN,—This patient has been sent here for an opinion as to whether surgery can relieve him. He is æt. 21 years, and comes with a large swelling in the neck. He says that no other member of the family has anything of the kind. Four days ago the first swelling occurred, and it was situated in the shoulder and neck, on the left side only. He experiences no difficulty in swallowing, and he has never lost his voice. The first point to observe is that the thyroid feels soft and natural on the right side compared with the left. The question is whether it is thyroidal in origin, or whether it is outside the thyroid. There are no other lumps anywhere else. The question of whether it is connected with the thyroid gland is not very easy to answer, because it is a localised swelling. It does not move when he swallows, and it does not follow the shape of either of the lobes of the thyroid. Still, for all that, it might have its origin in the left lateral lobe of the thyroid. Its chief tenseness appears to be just beneath the deep fascia. One does not notice any appearances of exophthalmos about the patient. His blood-vessels are pulsating and beating markedly there, and over the thyroid too, but I did not get any definite thrill in the thyroid. Has he got slightly increased fulness of the thyroid, such as many young men and young women have—that is to say a slight over-vascularity? It may be that there has been a sudden giving way of some vessels, and that there is hæmorrhage from the lower part of the lateral lobe of the thyroid. Of course it may be something quite outside the thyroid. One does not see any evidences of blood-staining in the skin as yet, but the mass is fairly deep, and therefore it may be a hæmorrhage in spite of the absence of discoloration. The patient seems not to have suffered general illness; if he had one might have thought it was an abscess in the gland. His tongue is somewhat coated. As the case is at present indefinite I think the best thing will be to ask Dr. Thomson, under whose care the patient is, to be good enough to get him to come up again

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in a fortnight's time. It is one of those cases which are more instructive if studied after an interval. At present I lean to the view that a hæmorrhage has taken place in connection with a somewhat turgid thyroid; that is to say, that it is a slight general hypertrophy of the thyroid, followed by local hæmorrhage.

My chief object to-day was to speak about a subject which is always before me, namely, some *abnormal conditions of the spine*. This is a very large subject, and if we were here two days a week for an hour each time for a year we should have plenty of material to last us that time. Still I hope I may say something which is of interest to you out of so much that there is to consider in connection with the spine.

We will begin by considering the *causation* of these conditions; in the consideration of lateral or other curvatures of the spine this important matter which is often left vague or kept out of sight altogether. Of course there are extreme cases which are quite definite enough. Here is one represented in a skiagram showing a little localised lateral deviation. The skiagram shows a dark rounded patch. That patch is a focus of tuberculosis in the spine and around. Thus tuberculosis may cause a lateral curvature of the spine. Here is another case which illustrates a condition that has already been put before the College:—*Congenital elevation of the scapula, or Sprengel's shoulder*, the subject being a boy who was under my care. This boy could not raise his arm above the horizontal position, and he was only discovered to be abnormal by the schoolmaster telling him to put his hand above his head, and the boy, appearing to refuse to do it, got a thrashing. This attracted the attention of the mother. I have had several such cases before, and one especially I studied closely. If we look at these skiagrams of another case of congenital elevation of the shoulder, we shall see that the chief part of this condition consists in a congenital lateral deviation of the spine. In that skiagram you will notice an alteration in the shape of the vertebræ. Some of the vertebræ appear to be fused together. And notice the interval there is between two of the ribs on the convex side. You will notice, also, what has not been previously pointed out, that one of the ribs is fenestrated; it is split, and forms a loop in that way. It is a very peculiar condition. No more can be done in such a case than to keep the patient healthy, and then, if there is no softening of bones, the deformity never gets very much worse. But I do not think much can be done to diminish the deformity in a severe congenital condition of that sort. The important point is not to direct energetic treatment to a condition which is really irremediable. The chief part of the deformity in these cases consists in a congenital lateral curvature of the spine, which, as it happens, is evidenced by elevation of the shoulder, and that feature has caused that name of congenital elevation of the shoulder to be given to it. I think it would be better to call it congenital localised lateral deviation of the spine with elevation of the shoulder. A more extreme degree of that same congenital abnormality is seen in "Willet and Walsham's" shoulder. In that there is actual synchondrosis between the vertical border of the scapula and the altered spinous processes of the altered vertebræ. Other exceptional cases are such as I show you now, namely, lateral curvature of the spine subsequent to empyema. It is that of a little boy who had an empyema opened a year or eighteen months before I saw him, and it had not then healed. It had been opened in the proper way, by removing part of one or two ribs, but still the discharge continued. I found that the lung had shrunk almost to nothing, and there was a great cavity in the pleura extending above the clavicle. I removed good pieces of four or five ribs, scraped out the cavity, irrigated it, and gradually it has closed up, and there is no more cavity left. But it leaves the boy with some lateral deviation of the spine. That lateral deviation, however, is diminishing with time, and as soon as conditions are favourable there I shall put him upon remedial exercises, which will still further diminish the deformity. Still another out-of-the-way cause of lateral curvature of the spine we see in nervous diseases. Here is one, for instance: a girl who had Graves' disease,—exophthalmic goitre—and you see well-marked lateral curvature of the ordinary type. Another nervous disease in which one sees these lateral curvatures of the spine is Friedreich's disease, which is commonly attended with deformities of the legs and feet only. But the spine very often suffers in Friedreich's disease. Another nervous affection is syringomyelia. In that the altered innervation leads to pronounced lateral curvature in many

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cases, and also to changes in the joints. Another condition which has come to my notice in which lateral curvature occurs—I am dismissing the less frequent ones to begin with—is the rapid growth that occurs on putting a patient with sporadic cretinism on thyroid treatment. In such a case the tissues grow so rapidly that the bone has not enough resisting power to bear the weight of the body properly, and in these circumstances it is not unusual to find lateral curvature of the spine. I want to emphasise the point that among those less common causes I include one which was given in most text-books on surgery as the most common, namely, an inequality in the length of the legs. A person may have the legs of unequal length, and the difference may be as much as an inch or so, and yet the person, if he is in good health, may have no lateral curvature of the spine. That is, of course, supposing that there is no rickets, or rheumatoid arthritis, or any bone-weakening condition. Still one sees inequality of legs cause lateral curvature not infrequently. One sees it in children who, from an early age, have had some hip trouble which has led to adduction. Such children, in order to uncross the legs, have to tilt the pelvis. If that dates from early life we shall have an adaptation lateral curvature of the spine. It is well to remember, then, that a short leg, if it begin early in life, will cause lateral curvature of the spine even in healthy people. In children who have bone weakness from rickets or gouty inheritance, that inequality of length of legs will very likely cause lateral curvature of the spine. But in their case it is rather the general pathological state that we must look upon as the cause than this immediately determining inequality.

Here is another boy. You see he has lateral curvature. There is one sharp curve to the right and another to the left. There is much lumbar lordosis. He has retraction of the sternum, but all this, Dr. James Taylor tells me, antedated his paralysis in the legs, and he has recovered remarkably, under Dr. Taylor's treatment, from most of the paralysis in the muscles of the leg. Notice how much this boy with his weakened muscles (and the abdominal muscles have shared in it) can do for himself in the way of temporary voluntary improvement. His weakness is, however, so great that it is not sufficient to leave him to his own devices. He has been much worse than he is now. I have devised a splint for him, and I may first call attention to one feature—the spring pelvic girdle, which fits him like a truss. We will look at the principles of this splint, because it is the most serviceable where an instrument is required in these cases of lateral curvature. This splint, the essential parts of which were introduced by Mr. Chance in 1850 at the City Orthopædic Hospital, differs from many of the apparatuses which are most commonly used in that it does not confine the ribs, but encourages their use in breathing. His ribs have sunk in; I expect this was due in part to rickets, since he still has thickening of the epiphyses. It makes a great deal of difference to this patient whether the ribs are left free or whether they are shut in by something like the common apparatus which is often advised, viz. the miscalled "poroplastic" jacket. The poroplastic corset made to fit to-day, in a growing person will be too small in a month's time. We are not quite perfect yet, and there are several things which are not yet in the current text-books, and this is one of them, namely, that these poroplastic jackets are incomprehensibly inapt, and unsuited to the conditions to which they are applied. The greatest care must be observed not to employ an instrument unless it is absolutely necessary. You can see a great deal of harm could be done by the improper use of such an arrangement as I show you on this boy. If he were left to wear this instrument without guidance, and to regard it as a means of salvation, *without exercises*, and if he were to wear it for ten or fifteen years without being made to periodically try to do without it, he would be much better without an instrument of any sort. The wearing of instruments is a thing which requires careful supervision by a medical man who is cognisant of the pathology of the condition that is being dealt with, and who will have it off twice a day for the purpose of having systematised exercises carried out. My practice is to have the names and particulars of these patients put down in a book, and on certain days they learn their exercises. Every one of these lateral curvature cases I have taught these exercises. The indications for those exercises I will discuss presently. This patient offered certain difficulties in fitting. I tried every type of instrument on him. One should always doubt one's own treatment, at any rate one should doubt its perfection, and be always ready to see if there is

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not something better. I therefore try every kind of instrument on him, including the poroplastic jacket. Here is a photograph of an instrument which is very commonly used. It is admirable as mechanism, but the upright at the back has a fixed curve and is made of tempered steel, so that the curve must be retained. And if there is an abnormal curve in the antero-posterior direction, as most of these patients have, it prevents the patient recovering to that extent; that is to say, the patient is prevented by such an apparatus from getting any better than he was when the instrument was first applied. But in this boy, wearing this apparatus, by altering the tension of the straps he can diminish the lordosis. And in many cases which have severe lordosis from any other cause, such as congenital dislocation of the hip, we can remove it all by degrees by the application of such an apparatus as this. All the curves which you see in the apparatus can be altered by means of a wrench. The great point about the apparatus is that everything is adjustable, and any improvement can be followed up by the surgeon. I do not want to lay stress on this more than is necessary; it is the best thing of its kind, in my own experience. I notice this same instrument is adopted by Mr. Watson Cheyne and Mr. Burghardt in their book on 'Surgical Treatment.' It supports parts which are tending to deviate, and the instrument does not allow the parts affected to move in a direction of increased deformity.

I now want to come to the common cases of the kind under consideration. How common this affection is I may illustrate by this. I have been going through a thousand cases of deformity, and I find that of this 1000 cases spinal affections account for 361 cases. The cases are taken exactly as they came. One source from which I took the figures was the North-West London Hospital, another was the City Orthopædic Hospital, and the third was my own private case-books. These are the results from three sources in three years, so you will see these spinal cases are extremely common, and it is more especially the common causes of deviation of the spine that I want to speak of to-day. But before going to the causes I want to say a word or two on the description of them. I have mentioned lateral curvature of the spine or scoliosis, and this term *scoliosis* I think a better term, because lateral curvature may be due to tuberculosis, whereas scoliosis proper cannot be due to that. By scoliosis we mean a non-tuberculous case. We have seen that there is a lordosis in the lumbar region; if we have a lordosis in the dorsal region, which is normally convex backwards, the first degree of lordosis will cause a flattening in the dorsal region. These flat back cases of lateral curvature are cases in which there is so much *dorsal lordosis*, and in some cases that lordosis in the dorsal region becomes a positive one, the dorsal region being concave backwards. Thus there may be a positive pronounced dorsal lordosis, and sometimes you can put your fist into the hollow of the back. It is not commonly taught that there is such a thing as dorsal lordosis, but it is very important because when it is present it means distinct encroachment upon the room of the heart and lungs in the chest, and it is in those cases that one finds very frequently a rapid pulse-rate and a very rapid breathing-rate, double or treble the normal rate. I have seen those cases referred to as hysteria and various grave chest affections, such as phthisis, because when the spine is twisted and the bronchi pushed aside you may hear tubular breathing or bronchial breathing in places which will surprise you. And if you do not happen to notice the shape of the spine the diagnosis of phthisis is not very unlikely, though the abnormal sound is really due to scoliosis. There is another commoner deviation, and that is increase of the normal roundness of the back, and that is called *dorsal kyphosis*. Sometimes it is called dorsal excurvation of the spine, but it is much better to speak definitely of it as a kyphosis. Although kyphosis is commonest in the dorsal region it can take place in the lumbar region, since the lumbar region is convex forwards, slight kyphosis leads to the straightening of the normal lumbar lordosis of the spine; thus in the lumbar region kyphosis may be present without any excurvation. Pronounced kyphosis in the lumbar region often is present in tuberculous cases, and in another class of case which I shall mention soon. I mention these because the causes of these conditions, which are very common, are the same. The same causes will lead to dorsal kyphosis as will lead to scoliosis, and the same thing will lead to lumbar lordosis which in another case will turn into lumbar kyphosis. So simply to say there is scoliosis or lateral curvature is to say nothing definite. Again,

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simply to say there is dorsal excurvation is no complete diagnosis. Here is a girl who has been under my care for some months. She has got very severe deformity, and she has been treated on what is generally recognised as the proper method. That is to say, lateral curvature of the spine has been diagnosed, and the teaching which is generally authoritatively put forward in this country that no instruments should be used, has been rigidly adhered to. Accordingly no instrument was used until she came under my treatment. It was felt that exercises ought to be used, and she was put under exercises. Therefore, she has been treated on thoroughly orthodox lines. Here is the result. I say there is something very much wanting in the modern teaching in this subject—how to deal with a lateral curvature of the spine. Here the doctor has done what the highest expression of professional opinion taught him. If this girl had had an instrument such as I have shown you when it was found the exercises were not arresting the deformity, I have no doubt she would have been much less deformed than she is now. My rule in reference to this condition is—I give the patient a chance to get well without an instrument, but if I see he is getting worse in spite of that, then, in addition to exercising and dieting, I design for them an instrument. This I take to be a rational form of treatment. I quite understand the prejudice against instruments, because I have seen instances in which this particular instrument has been abused; patients have been allowed to go on wearing it for years when they really only required it a month or two. But when the bones are softening and the muscles are undergoing gouty contracture, then is the time that some support is required without making the patients absolute invalids, putting them to bed, and so weakening the general health. Such patients want so much support as to enable the bones to carry on their function.

How shall we describe this girl's deformity? There is, in the dorsal region, a curve convex to the right, therefore there is right convex dorsal scoliosis. In the lumbar region there is convexity to the left. She has thus left-convex lumbar scoliosis. We have then to look at the profile to see whether there are any antero-posterior deviations. We notice she is very flat in this part of the spine, but I think that is accounted for by the rotation. In her case there is not so very much alteration in the antero-posterior curves, therefore we can fairly completely describe her case as right-convex dorsal *plus* left-convex lumbar scoliosis. That is simply the anatomical diagnosis. Let us see if we can make a pathological diagnosis. Very often in these growing children, even up to fourteen or fifteen years of age, one finds a distinct enlargement of the epiphyses. I am of opinion that about one third of the cases of lateral curvature of the spine are definitely rachitic, and this applies to private equally with hospital practice. In one work on the spine which I have read it is stated that rickets is a disease which occurs only in the families of the extremely poor, and in the very young members of those families. But as a matter of fact, it occurs in the families of the very rich, it occurs in the families of the middle classes, and it occurs during the period of growth at all ages. To speak of rickets as occurring only in the very poor is to show a misconception of the whole pathology of lateral curvature.

I have boldly stated that rickets accounts for about one third of the cases of ordinary scoliosis, and I think I shall find that is the case when I have finished the analysis of my cases. Rickets is an important factor in the causation of scoliosis.

A second important group is the *osteo-arthritic*. I now show you another girl, rather an earlier case than the one we have just seen, and we see she has some increase of the dorsal convexity backwards—there is dorsal kyphosis and some increase in the lumbar lordosis. We see that the lateral curvature is not great. There is no difference in the length of the legs. There is a projection of the spine at the root of the neck, and another at the junction of the dorsal and lumbar regions of the spine. She has had the tonsils removed, and has suffered much from sore-throat. This is an *osteo-arthritic* case. Note also the localised thickening at the articular end of one of the radii. We have these localised weakenings of the spine in her case, and we have got that kyphosis associated with a tendency to chronic, and probably acute exacerbations of chronic inflammatory conditions of the tonsils and their neighbourhood.

That brings me to the second great cause of these cases of deviation from the normal in the spine. I would have had more of them to show you, but most of them are young women in service

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who have not very much the matter with them, and it is rather a trying ordeal for them to be shown. I have copied the notes of a case, that of L. E—, æt. 27, a servant. She had dorsal kyphosis like this one, and I ascribed it to osteo-arthritis. She had also a pigeon breast, which has developed in the last year or two. She had pain in the back, which has been worse during the last twelve months. She has had pains in her back for years, and it was chiefly in the lumbar and sacral regions, and is said to have dated from a fall six years ago. The sterno-clavicular junctions project forwards on both sides, especially on the right side. The patient says that is a recent phenomenon. At one time the patient could not bear her clothes against certain portions of the spine. Her bowels and menses have been regular. The mother suffers much from "rheumatism," and has her hands deformed. The father is healthy. One brother died of phthisis at the age of fifteen. This case illustrates a point which is also shown by the last patient I showed you, namely, how patchy osteo-arthritic lesions are apt to be. There are two foci in the spine, and there was one in the hand. Rickets affects the patient more generally; rheumatoid arthritis is more patchy as a rule. She had quinsy and sore-throat at times, but not more than most people, she thinks. I ordered her breathing exercises and other exercises, such as lying down and getting into the sitting posture without using her hands. Another useful exercise is lying face downwards and arms extended out, and simultaneously arching the neck and body backwards and supinating the arms. This exercise is particularly useful in dorsal kyphosis. I shall afterwards add rotation movements. I begin always with breathing exercises; they serve to extend the dorsal spine, and made the patient acquire a habit of taking in more oxygen, which is needed for all metabolic processes, and is especially called for in rickets and rheumatoid arthritis. Then I gave her powdered guaiacum, five grains twice a day after meals, and ordered her to drink more milk and to take all bread in the form of toast. That is all I have done in the way of internal treatment. That was on November 26th last year. In January I have a note, "much pain between the shoulders, but the old lumbo-sacral pain is less." Yesterday I made a note that she has been free from pain for the last month, and that is the first time she has been free from pain for years. The instrument has brought her nearer to the normal form, and the antero-posterior curve has practically disappeared, so that the bones are bearing pressure where nature intended them to. With the improved shape of her spine the pain has all gone. That is one of hundreds of cases I could tell you of, and many of them are servants earning their living, which they manage to continue doing by wearing the instrument; otherwise they would lose their work. After the spine has been fixed in an abnormal position sufficiently long it becomes more and more difficult to set it right—the deformity becomes fixed. "Arthritism" accounts for another third of the cases of curvature of the spine. What do we mean by "rheumatoid arthritis?" Sometimes it is very severe, affecting every joint, and associated with intense anæmia. Moderate cases such as I have described and shown you to-day are most of them associated with feeble digestion, and, as far as my little medical knowledge goes, guaiacum is good for that. If the digestion is very much out of order I call in the aid of a physician.

This leaves about another third of the cases to account for. Many of those, I believe, are due to temporary disturbances of digestion, leading to hyperacidity of the tissues. We see the patient when that condition has all disappeared, like the first girl I showed you this afternoon. She is practically quite healthy now, and she should be upright. Possibly the pathological condition which caused her deformity to come on only acted for two or three months; the cause has ceased to act. She has now a few years in which to grow, and that is why I have put on this instrument; the only hope she has of improvement is in the direction of her growth. The latest professional statement on the subject of scoliosis is contained in a report of the committee of the Clinical Society. At the time it came out I have no doubt it was absolutely abreast of the times, but it is now many years old, and I hope the Clinical Society will before long publish another report, because I think it is time something was added to it. It says there that most of these cases appear to be associated with "some delicacy." I have ventured to suggest to-day what that delicacy is. In many cases it is rickets, in many it is goutiness, or, at any rate, a disturbance of digestion associated with osteo-arthritic lesions. And these cases, when watched, often look as

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they were perverted hysterical conditions. Some of these cases which at first sight have appeared to me to be hysterical have ultimately turned out to be those dreadful cases which become rigid in the whole length of the spine. If I had treated them on the idea that they were hysterical only, I should have been dealing unjustly with a most serious pathological condition, and in these spine cases we cannot be too careful about talking of hysteria. True there are some which are really hysterical, but most of the cases which are so called turn out to be neuro-osteo-arthritic cases, because where there is goutiness in the body the nerves are the first to feel it. These patients cannot help their oddness of posture. They are not very ill; they seem whimsical about their feeding, and are generally trying and irritable. There is this common point in the gouty and rachitic cases—they are both associated with lack of oxygenation. What suits rickets suits goutiness, and there may be a common element in the two things. Can we generalise as to the nature of these gouty deformities in the spine? I think we can. I do not know if any of you have watched a hammer-toe develop; I have watched it several times. It began in myself once, when I got too much Londonised, and went without a holiday. My toe dropped, and I could not straighten it. I have had the condition in boys intended for the Navy in the same stage. There is no paralysis about it, but, do what they will, they cannot straighten the toe. One puts a little splint on it which they can wear inside their stocking. This restores the proper "average form" to the toe, and gradually the extensor muscle resumes its work. The original deformity is due to a kind of reflex inhibition of the muscle. You can in every case of acquired hammer-toe absolutely certainly prevent the need for operation if you get the case before any shrinkage of ligaments takes place. If once this shrinkage has taken place you cannot get a hammer-toe straight without an operation. These osteo-arthritic curves in the spine are very much the same as that of hammer-toe. If you get them early you can treat them by holding them gently in a suitable light instrument, combining that with such exercises as the severity of the case makes the patient capable of undergoing. But if that deformity is allowed to go untreated for a certain length of time there will never be complete cure. It is upon the catching of these cases early that success in treatment depends. It is a common experience to get a crick in the back from want of fresh air and exercise. But if the victim goes into the country, and walks perhaps ten or twenty miles, that crick goes off. These patients, as we get them, have gone beyond that stage, the capillaries have formed new offshoots, and there is new inflammatory tissues in those bones. The patients cannot "walk it off," and they cannot help the condition at all. You have to treat them humanely and rationally, alter their diet, get them out into the fresh air, and have them rubbed and exercised, not in a general way with dumb-bells or what not, but by exercises appropriate to their pathological state. They must be watched; and if they do not get better with these measures alone, then give them support *in addition to the other treatment.* That is what this instrument enables one to do without injury to the patient's health. If you neglect support, there will come a time when you can no longer hold the patient in a better position by means of the hands, or by means of an instrument. The deformity has become *fixed.*

Time does not allow me to go into another aspect of these cases—a very important one,—the relation between abnormal conditions of the nasal passages and throat and abnormal spinal curvature. On these matters I hope to speak another time.

Pathology of Tuberculosis.—Some points brought out by Black are the difference of the tubercle bacilli from others of the acid-fat types; the possibility of the tubercle bacillus being a phase in the development of a fungus, as held by Metchnikoff; the distinction between bovine and human tuberculosis, which he considers leaving much to be said; and the question of how infection enters the system. He thinks hereditary tuberculosis, though positively shown, is negligible on account of its rarity. The question whether tuberculosis can be acquired through ingesta is also of importance, but he has never yet seen a case of primary intestinal tuberculosis in several thousand autopsies. Inoculation tuberculosis is comparatively frequent, and he holds that at least 50 per cent. of those that are dying of the disease show evidence of having suffered from it.—*Journ. A. M. A.*, July 12th, 1902.

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THREE CLINICAL LECTURES
ON
THE DIAGNOSIS AND TREATMENT OF RECTAL DISEASES.

Delivered at the Medical Graduates' College and Polyclinic, May 26th, June 2nd and 9th, 1902.

By F. C. WALLIS, F.R.C.S.,
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LECTURE III.

NON-MALIGNANT STRICTURE OF THE RECTUM.

As might be inferred, the common result of the progressive ulceration described in my last lecture is a fibrous stricture, and this ulceration is probably the common cause of non-malignant stricture. Various other causes have been given, but a large number are usually classed as unassigned. These are probably all due to septic ulceration. A certain percentage which are said to be due to tertiary ulceration must be received with caution, as in most of these cases no history of the primary disease can be produced. Moreover, there is no response to the usual remedies. This statement of mine will probably be received with a certain amount of scepticism, because of the prejudice of many years during which each book on this subject has reproduced the old stereotyped list of causes. My statement as to the frequency of sepsis causing these fibrous strictures of the rectum is founded on facts which are difficult to disprove. Moreover, causes unassigned do not appeal to me. Such a formidable result of disease as fibrous stricture is cannot be classed among those diseases which, for want of knowledge, are described as idiopathic in origin. From whatever cause arising, a fibrous stricture is the result of previous inflammation, which, whether associated with ulceration or not, yet by the ultimate production of fibrous tissue, the invariable end of all chronic inflammation, causes stricture. That such a stricture may occur without any ulceration of the mucous membrane has been shown at various times by cases, always in women, where inflammation in the neighbourhood of the pelvis has been associated with stenosis, and when the levatores ani muscles spasmodically contract, a stricture occurs, in addition to a certain amount of stenosis which is always present. These strictures as a rule are not of sufficient inconvenience to call for any strong measures of treatment. In examining such a case, it will be found often that what appears a very obvious stricture on ordinary examination will almost entirely disappear if the patient is examined under an anæsthetic. Or, if this is not desirable, a moderate-sized conical bougie is introduced for a minute; and then if a digital examination is made, the muscular spasm will be found to be overcome by the bougie, and the amount of stricture apart from muscular spasm is arived at. This is a matter important to remember when dealing with contractions which are due to pre-existing inflammation outside the rectum.

When a fibrous stricture of the rectum is fully established the condition of the patients is typical and forlorn. They are emaciated, have frequent attacks of pyrexia, intermittent diarrhœa and constipation, and extremely bad digestion. Unless these patients are in some way relieved they become so seriously ill and their local condition complicated with fistulæ, etc., that they gradually fall into a condition of chronic pyrexia and so die. The condition under which these patients live is well shown in the following case of a woman who is now under my care. She is aged thirty-six, and came originally with an annular stricture three and a half inches from the external sphincter. This had been in existence for some years, being the ultimate result of a protracted labour associated with a septic discharge. Her condition when she came under my care was as follows:

Owing to the great difficulty caused by the stricture aperients were taken every night, the effects of these lasting until the following midday. During the morning the aperients would act from ten to a dozen times. The rest of the day, although somewhat exhausted, she was fairly comfortable, but unable to do much. If the bowels did not act every day, the woman became quite ill and suffered considerable pain. The whole of her existence was practically given up to the business of getting rid of the contents of the intestine.

Another case of a somewhat similar character was that of a woman, L. T—, æt. 40, married, five children. Her past history was that eight years ago, when the third child was born, she had a bad confinement. For the last six years there had been increasing difficulty in getting the bowels to

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act, and frequently during this time she passed blood. She had been in various hospitals for stricture of the rectum, which was temporarily relieved by bougies. When seen by me at St. Mark's Hospital in February, 1899, the rectum was ulcerated as far as one could feel, and about four inches up was a tight stricture which did not admit the tip of the forefinger. The smallest sized bougie was passed with difficulty, and gradually the stricture was dilated up to a No. 6 bougie.

These two cases show the condition under which these patients exist, and it will be admitted that every possible means should be used that relief shall be obtained.

Treatment.—The palliative treatment of fibrous stricture is by passing bougies. The relief by this means is quite evanescent in character, and has the disadvantage of being extremely painful at the time, and it is only when the patients refuse to have anything else done that I should advocate their use except as a temporary measure. In passing a rectal bougie care must be taken not to use any force. At the place where the stricture is and where the bougie will be tightly held there is often some ulceration and weakening of the bowel, and it is quite easy when misapplied force is used for the bougie to pass through the rectal wall into the tissues beyond, possibly into the peritoneal cavity. When an obstruction is arrived at on passing the bougie, gentle pressure should be maintained, and the patient should be requested to strain on to the bougie. By these means the muscles are contracted and pull on the stricture, and thus may possibly be of some use in helping the bougie to pass. Another precaution which should be taken is to have the ulcerated surface cleansed as much as possible before passing the bougie, so that this may not be a means of spreading the septic condition still further up the bowel.

Operative treatment in these cases consists of linear incisions and dilatation of the stricture, posterior proctotomy, excision of the stricture, with or without previous colotomy, and finally, when all of these are of no avail, colotomy is often the greatest boon to these patients. The linear incision and dilatation give temporary relief only, and the stricture in course of time returns again as bad if not worse than before.

Posterior proctotomy is effective for a while also, in that by this means the stricture itself is divided and the contents of the intestine are enabled to escape without any obstruction, but gradually this incision closes, and the strictured condition appears again with a return of all the old symptoms. The ideal treatment in these cases is excision of the stricture, bringing down healthy mucous membrane to the margin of the sphincter. This operation I have already described, and I will only add that treatment by excision, although difficult and not unassociated with danger, should be done much more frequently in these cases than has yet been the custom. As an instance of this I quote a case which has already been reported, of a young healthy-looking girl of eighteen who, as the result of prolonged septic ulceration of the rectum which had been noticed since the age of five, had a marked stricture between three and four inches within the bowel. Excision was performed on her without a previous colotomy, and the result to-day is that she is in perfect health three years after the operation; there is no stricture, no contraction, and she has been earning a livelihood since she left the hospital in July, 1899. This patient had after the operation a considerable amount of purulent discharge, owing to the fæcal infection of the part. Since that time I have always preceded this operation with a temporary colotomy. The result as far as benefit goes, and quickness in healing, is well illustrated in the further history of the case which I referred to just now (L. T—). This woman had colotomy done in April, 1899. For some reason she left the hospital a few weeks afterwards, and attended as an out-patient until November, when she was readmitted and the stricture excised. The subsequent course here was uneventful as, owing to the colotomy wound, the local area of operation could be kept quite clean, and an uninterrupted recovery was the result. A month later the colotomy wound was closed in the manner already described, and she left the hospital in the best of health, and has been seen at various times since looking ten years younger and without an ache or a pain, a different picture to the thin, miserable, pain-stricken creature she was before the operation.

Such cases as these warrant one in saying that where possible this operation of complete extirpation of the stricture should be performed. When the stricture is so high up or so prolonged, or when the destruction and ultimate fusion of the

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tissues has been so severe as to render it impossible to do this operation colotomy is by far the best, safest, and most effective treatment, and the patients upon whom I have performed colotomy, because of this state of affairs, have soon become accustomed to it, and the improvement in health has been most striking. Moreover, through the colotomy wound the lower and affected area can be treated by irrigation, etc., and there is still the off-chance, when colotomy has allowed all the local inflammation to settle down, that something in the way of a permanent cure may yet be done. If these people are content with a colotomy, it is wise perhaps to remain contented also. But should the patient desire that an attempt should be made to re-assume as far as possible the normal state of affairs, some special operation will have to be done, such as that which was published some years ago by Sir F. Treves in his case of idiopathic dilatation of the colon.

Neoplasms of the Rectum.

These are divided, as in other growths, into innocent and malignant. The various innocent growths of the rectum are much like those of other portions of the mucous membrane. They consist of polypi, of which there are two main varieties: one being composed mainly of glandular tissue, and the other of fibrous tissue covered with mucous membrane. Both these forms are attached to the bowel by a pedicle, slight in character and occasionally rather long. Commonly found in children, they may also occur later in life. They do not give rise to any very marked symptoms, and exist for years without the patient being aware of their presence. Usually single, but two or three may occur in the same individual. When attention is called to them it is generally from a feeling of having some irritating substance in the bowel, or some loss of blood, especially in the adenoid variety, or, finally, the polypus may be sufficiently near the sphincter or the pedicle long enough to cause the tumour to appear outside. The growth is easily felt on examination, and when not too high up may be summarily dealt with at the time of the examination. It is a smooth, rounded tumour, not larger, as a rule, than a large cherry, and the pedicle can be traced back to the mucous membrane. Should the patient be complacent and the polypus situated so that the pedicle is not too high up, by a little manipulation the growth can be brought outside and a ligature placed round the pedicle, after which the polypus can be removed, and a cure is thus effected. But the difficulty of this method occurs when, possibly owing to the frailty of the pedicle, this tears through before the ligature has been applied, in which case some hæmorrhage may occur. If, therefore, on examination the pedicle is attached somewhat high up or is very thin it is better to wait until the patient can be given an anæsthetic, when, after dilatation of the sphincter, the polypus can be easily seen, secured, and removed. Again, where there may be more than one an anæsthetic is desirable. After-treatment in this case is quite unnecessary. The pedicle retracts, and the ligature comes away in the course of a day or two, and no further trouble is felt in connection with it. A form of polypoid growth which is pathologically of an innocent character yet clinically often gives rise to serious symptoms is that known as multiple polypi. This condition is a rare one with which it is practically impossible to cope except by palliative measures, inasmuch as these multiple polypi may exist over the whole of the large intestine. I have had two such cases under my care, both of which occurred in boys. The symptoms they complained of were loss of blood mainly, and a certain amount of straining at times. Neither of these symptoms were persistent. The less this condition is interfered with, and the healthier the patient can be kept, especially with reference to digestion and the contents of the intestine, the better. Should hæmorrhage occur, small doses of tincture of hammamelis may be given for a time with good effect, and constipation should be avoided by simple remedies. Occasionally severe hæmorrhage occurs in these cases, and it may be necessary even to transfuse in certain instances.

Villous tumour of the rectum is of a borderland character from a pathological aspect, and of rare occurrence in this region. It is usually attached by a broad base to the mucous membrane of the bowel, and may exist for a considerable time and grow to a large size without causing the patient any great amount of inconvenience. The following case is an example of this.

A healthy-looking woman æt. 35 was sent to me at St. Mark's with a history of considerable loss of blood from the bowel, and a quantity of blood-

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stained discharge. On examination a large growth was found in the anterior wall, which sprang from a broad pedicle and then exfoliated, making quite a large mass. The patient was admitted, and anæsthetised, and the growth pulled down after stretching the sphincters. A stout ligature in a pedicle needle was used to transfix and tie the base, the main portion of the growth being removed with scissors. The patient made an excellent recovery, and left the hospital within three weeks. She has been seen several times since, and the transfixed pedicle is now gradually disappearing, but for some months it was a source of anxiety, and showed no signs of disappearance.

The microscopic characters of the tumour were reported on by two experts. The one pronounced it probably malignant and the other probably not; and this about represents the pathological situation of the growth. Clinically, although the base is hard and quite fibrous, yet after removal these growths do not, as a rule, return. Their main symptom is loss of blood, and on examination a very large mass may be felt which in many ways feels like a malignant growth; but the base, although broad, is not infiltrating, and the mucous membrane can definitely be felt coming off the bowel and spreading over the commencement of the growth. But these cases are sufficiently rare to allow of a mistake being permissable. The only suggestion is that a guarded diagnosis should be made, and the patient should not then and there be condemned without some further evidence of malignant disease.

Malignant growths of the rectum are unfortunately common enough, and occur at all ages, although more common in advanced adult life than any other, but it must not be forgotten that carcinoma of the rectum often occurs at quite an early age. The treatment differs very much if the growth is in a young or old individual. I have seen patients with this disease at fifteen years of age, twenty-three, twenty-eight, thirty-three, and so on, and the younger the patient the more rapid the growth, and the more hopeless the idea of any operative measure; whereas, when the disease commences late in life its growth is slow, the symptoms often are practically unnoticed, and even after the existence of the carcinoma for some months great benefit is derived from operation. The fact that in these cases of carcinoma in adults the symptoms are so slight and so vague is, in itself, often the cause of the patient wasting valuable time as far as operative measures are concerned, because neither he nor his medical man, if he has consulted one, consider the symptoms of sufficient moment to suggest even such a condition as carcinoma. In the young subject of carcinoma the symptoms are often sufficiently severe at an early period to call for an examination. They are pain and loss of blood, and possibly some difficulty in the action of the bowels, yet these three symptoms are the common signs of hæmorrhoids. I can only repeat with renewed emphasis what I said in the first lecture with regard to the absolute obligation of insisting upon making a thorough rectal examination in all such cases.

Taking the case of carcinoma in a young subject first, when the patient complains of the symptoms which I have just mentioned, the obvious custom should be to make no diagnosis until the cause of the symptoms complained of has been definitely discovered. On examining such a patient when the growth is within reach of the finger, the irregularity and hardness of the raised margin, the want of any suggestion of a pedicle, and the ulcerated condition of the more central portion of the growth, are sufficient clinical signs to make a diagnosis. No other growth has all these. I would particularly lay stress upon the induration as being against the case being hæmorrhoids, and the raised margin which is indurated would prevent any mistake as to the nature of the ulceration. The want of a pedicle of any sort prevents the mistake of calling it either a polypus or a papilloma.

The treatment of these cases is most unsatisfactory, even when the growth admits of complete local removal. The rapidity of the growth is in conformity with the youthfulness and growth of the tissues of the young subject, and has very early infiltrated far beyond the apparent limit. Almost before the original wound has healed the growth returns locally or in the neighbouring tissues, and the remaining treatment resolves itself into various palliative measures until such signs of obstruction arise as will necessitate colotomy being performed to prevent intestinal obstruction. This is a melancholy picture which I hope may be painted in brighter colours in the course of a few years. Turning now to this condition when it occurs after

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middle life, when seen and recognised sufficiently early much can be done.

So much impressed have I been by the paucity of symptoms in these cases, that the tendency in my own mind is always to take a grave view of a patient past middle life who is otherwise healthy, and complains only of what may be termed causeless diarrhœa, to which is added some slight loss of blood. A large number of patients from the country come to St. Mark's with merely these symptoms, and when questioned as to whether the loss of weight has been noticed, if the answer is in the affirmative the diarrhœa is attributed as a cause. Under such circumstances, even although no growth can be felt, it is a mistake to definitely say that no growth exists. As illustrating this point, I will briefly detail the history of a patient sent me recently by Dr. Brewer.

This man, past sixty years of age, was in every way healthy except that he complained of an occasional slight loss of blood, a little difficulty in getting the bowels to act, and occasional attacks of diarrhœa. On examining by the rectum there was nothing to be felt. On abdominal palpation the same negative conditions prevailed. I would like here to give a word of warning to the possibility and frequency of mistakes being made by finding some sort of swelling when examining the left iliac region. In a thin patient the sigmoid may very easily be felt, and sometimes its contents do rather present a tumour. But unless the same swelling is noticed in the same place on two or three different examinations, and the consistency of it is sufficiently hard, no positive diagnosis of a growth should be given. In the instance at present under consideration, as I have said, nothing could be felt, and yet so convinced was I of the probabilities of a new growth being present somewhere in the sigmoid, that I did not hesitate to advise an abdominal incision with a view to diagnosis, and any possible treatment which should be considered advisable if the growth were discovered. After thinking it over for nearly a month, and as the symptoms continued, the patient at last reluctantly agreed to an operation.

An incision was made as for a colotomy, and when the abdomen was opened, on examining the sigmoid I traced it down to a point just over the brim of the pelvis, where a small, hard mass involved most of the circumference of the bowel and fixed it immovably to the side of the sacrum. The site of the growth and the extreme fixity, together with the advanced age of the patient, made any idea of removal out of the question, and a colotomy was then performed. The patient made an excellent recovery and has been relieved of whatever pain he suffered, and further intestinal obstruction, which was certain to occur before, has now been obviated.

This is one instance of a very large number which I have seen at St. Mark's and elsewhere, where carcinoma of the rectum has been present without causing any severe symptoms.

Another case of equal interest was one which was sent to me by Dr. Roberts, of Deal. The patient was a particularly healthy Channel pilot, and was sent up because of a painful fissure. This being quite apparent on inspection no further examination was made until the patient was under an anæsthetic, when the fissure being divided and a digital examination made higher up, to my surprise and dismay I discovered a large growth involving the whole of the rectum and quite beyond the reach of operation. This patient had not complained of any pain referable to this, had not noticed any loss of blood, and was particularly robust and well coloured. He made a good recovery from the fissure and returned home. I saw him some months later, when he was gradually feeling the effects of the growth, but the need for colotomy did not arise.

Sufficient, I think, has been said to show that carcinoma may exist in the rectum with slight symptoms, and I hope I may have made it clear as to the obvious duty of making every possible examination in these cases with slight symptoms. On examining a carcinoma in a patient of this age the margin will be found raised and quite indurated, and the general consistency of the growth is much harder than in the case of younger people. The tendency is to encircle the bowel, undermining the epithelium, which is raised, and finally ulcerates. The question of removal depends upon the fixity of the growth, and to a less degree upon the extent. The best cases for removal are those which are limited to one portion or other of the bowel, which do not involve the sphincter area, and the upper limit of which can be reached by the examining finger. If the growth is not fixed it can be thoroughly removed, and the probability of

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recurrence if this is effectively done is less than in any other form of cancer, except perhaps in cases of epithelioma of the lip.

As to the question of fixity, a growth may, under certain conditions of contraction of the pelvic muscle, appear to be fixed, whereas when the patient is examined under an anæsthetic and the muscles are relaxed the growth may be found freely movable. This is well instanced in the following case, which I have recently operated on at St. Mark's:

A man æt. 57 came to the out-patients with the symptoms which I have already mentioned. On examination a growth limited to the anterior wall was felt. It was obviously slow growing, and the upper margin could be easily reached, but it felt so immovable that both I and others who examined it were extremely doubtful as to the possibility of removing the growth from the bladder, and it was therefore put to the patient that he should give permission for colotomy should the necessity arise. This, however, he absolutely refused to do, and it was agreed that an anæsthetic should be administered, and that, dependent upon the examination under these circumstances, I would or would not operate. I found that when the muscles were relaxed the growth was not adherent, but moved freely over the bladder. It was removed, and the patient has made a good recovery. This case well illustrates the possible effect of muscle contraction in altering the clinical aspects of a growth. It also instances the best class of case for operation. Here no division of the sphincters is necessary, nor any such formidable operation as division and removal of a portion of the sacrum is required.

When the growth encircles the bowel and is so high that the upper limit can scarcely be reached, if at all, then the sacral operation is the best. And here an important question arises as to what are operable or inoperable cases. It is a question which has not yet been decided. The tendency is now to do far more than was formerly done, and the results warrant the action. The blank wall of despair with which a patient is faced when he is informed that nothing can be done is a matter for consideration, and it is an open question as to whether an attempt should not be made in a larger number of cases where there is even only a forlorn hope of removing the growth. The mental condition of these patients is a much brighter one than if they know that nothing further can be done. Moreover, in a certain number of cases when the growth has returned it has not been in the bowel itself, but in some neighbouring part, so that they get whatever period of relief is afforded to these people without suffering those distressing symptoms that occur at the end.

Another point of some importance is the question of the advisability, when an excision of the rectum is to be performed, of doing a preliminary colotomy. The points against it are that it means three operations instead of one. The advantages are that after the colotomy the growth previous to the operation for its removal as well as the bowel below the colotomy can be thoroughly irrigated and rendered as clean as the circumstances permit. At the time of the operation all fear of fæcal infection is done away with, the peritoneum can be opened with impunity, and much more of the bowel can be removed under such circumstances. And best of all the subsequent healing is allowed to go on without any fæcal contact, with no *muscular straining*, and, in fact, under the best circumstances for complete and perfect wound union.

Ten days should elapse between the colotomy and the excision, and a fortnight to three weeks should be allowed after the excision before closing the colotomy. The details of the sacral operation need not be gone into here. When a case is hopeless as far as local removal is concerned it is better that the colotomy should be performed too soon than too late, and when once the natural repugnance of the operation has been overcome the patients can keep themselves free from pain for a considerable time, septic absorption from the growth may be averted by washing through, and the rapidity of the growth is considerably retarded in many cases.

Rheumatism of the Nose.—Frudenthal describes a condition of inflammation of the turbinates in connection with rheumatic arthritis elsewhere, and reports several cases, a number of which he has seen. He thinks many cases of what are called simple coryza may be rheumatic, and sees no reason why the nose should not be affected just because the articulations there are not as typical as those in other parts of the body.—*Journ. A. M. A.*, July 12th, 1902.

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TWO CLINICAL LECTURES

ON

"UTERINE DISPLACEMENTS."

Delivered at the Medical Graduates' College and Polyclinic,

By W. J. GOW, M.D.,

Physician Accoucheur to Out-patients, St. Mary's Hospital, and Physician to Queen Charlotte's Lying-in Hospital.

LECTURE I.

GENTLEMEN,—I have decided to offer a few remarks on the subject of uterine displacements, a subject which is admittedly not a very easy one to deal with, and to some extent also a contentious one. But the fact that uterine displacements are so often met with must serve as an excuse. As you are aware, there is nothing more common than for a woman to seek advice from a doctor because, as she thinks, her womb comes down. Sometimes she may be correct in her diagnosis, but it is generally wise to inquire whether anything actually comes outside, because unless something does come outside, it is obviously impossible for her to speak with any certainty on the matter, and therefore her statement should be taken with caution. In many cases where women make such a statement they will admit that nothing comes outside. One therefore asks the woman, "How do you know that your womb comes down?" Her answer is generally that she has a bearing-down sensation, and in many cases where women suffer from this symptom they translate this bearing-down sensation into an actual descent of the womb.

I may say, therefore, at the outset, that a bearing-down sensation is often only a sensation, and that discomfort in the pelvic organs often expresses itself to a woman as a feeling of something coming down. And it is well to remember that when a women feels as if something is coming down it does not necessarily mean that anything comes

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down at all. Of course, in many of those cases where that sensation is present there is some real protrusion or descent.

There is some difficulty in classifying the varieties of descent, and in the English text-books you will find that very little attempt has been made to do this. However, I think the simplest way of classifying the commoner forms of prolapse is to divide them into two main kinds, to which there will be subsidiary kinds. The first is the vagino-uterine descent, and the second, utero-vaginal descent. By vagino-uterine descent I mean those cases in which the vagina comes first and leads the way; sometimes, and in most cases, the anterior vaginal wall leads the way, but in other cases it is the posterior vaginal wall which comes first. When the anterior vaginal wall leads the way it produces that commonest of all protrusion, the cystocele, of which I shall have more to say directly. The anterior vaginal wall having descended, the cervix, to which the anterior vaginal wall is attached, becomes dragged downwards, and the uterus with it. Sometimes considerable resistance is offered to this descent by the utero-sacral ligaments, which are attached to the uterus at the level of the os internum, and as a consequence the supra-vaginal cervix gets pulled out and elongated, and this condition of affairs is observed in nearly all cases of considerable vagino-uterine descent. The greater the degree of resistance offered by the sacro-uterine ligaments to the descent of the uterus, the greater will be the elongation of the supra-vaginal cervix. In the case of utero-vaginal descent, on the other hand, it is the uterus which comes first; the uterus sinks down owing to the relaxation of its ligaments, and as it descends gradually into the vagina it everts both the anterior and posterior wall equally. In the former variety one of the walls of the vagina was completely everted before the cervix appeared at the vulval orifice. In the case of utero-vaginal descent, the uterus gradually everts both walls of the vagina equally from above downwards, and thus the cervix gradually emerges at the vaginal orifice. The ultimate result is much the same, but there is a difference, which we shall see in a moment. In addition to those two main kinds there is another state of affairs which is often overlooked. It is a rare condition, which may be associated with complete eversion of both vaginal walls and the appearance of the cervix externally. This is the condition known as vaginal enterocele, where, in other words, a hernial protrusion of the intestine takes place, pushing the upper part of the posterior vaginal wall downwards, and ultimately through the vaginal orifice. With that protrusion the uterus and the rest of the vagina is almost always carried down as well. I shall have to say more about this soon. In this case, when both vaginal walls and the uterus are extruded, you will find on examining the procident mass that there are coils of intestine in the pouch of Douglas, a thing which is never observed in ordinary cases of procidentia.

With regard to vagino-uterine descent, that is the commonest form of descent; and it is very rarely met with except in women who have borne children. It is primarily the result of weakness of the pelvic floor. This, of course, is very frequently observed after childbirth, owing to the stretching and laceration of structures forming this part which have occurred during this process. It is important to remember that childbirth is frequently followed by a state of imperfect involution. We are all familiar with subinvolution of the uterus. It is a term used to cover a variety of conditions met with after delivery in which the uterine body remains larger than it should be. But it is often forgotten that there are other parts which have to undergo involution besides the uterus; for instance, the vagina and the abdominal walls. I want you to understand by subinvolution of the vagina a condition where the vagina has not become restored to its normal condition after childbirth, a condition which is extremely common, and which you can see every day if you look for it. That condition is a very powerful predisposing cause of vagino-uterine descent, and tends to produce a cystocele or rectocele. The walls of the vagina, instead of contracting up and regaining their normal tonicity, remain loose and lax, and if the intra-abdominal pressure is considerable, protrusion of the lax vaginal walls may readily occur.

You are equally familiar with the fact that the abdominal wall fails frequently to undergo involution; and if this occurs in an exaggerated form, it will give rise to the condition known as pendulous belly. This condition of imperfect involution of the abdominal walls is a fruitful source of discomfort, and indirectly plays a part in the formation of these prolapses by failing to adequately

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support the abdominal contents. Extra strain is thrown on the pelvic floor, and thus extrusion of the parts may result. Subinvolution of the vagina represents a form of weakness of the pelvic floor which precedes vagino-uterine prolapse. There is another factor, namely the absence of the perineal body. The word perineum is a term applied to two things: it is applied properly to the perineal body, which is a mass of muscular and tendinous tissue between the lower part of the vagina and the rectum. But the term is also applied to the skin which extends from the anus behind to the fourchette in front. The perineum strictly means the perineal body. The destruction of this triangular mass of tissue is extremely common during the act of childbirth. To what extent does the loss of the perineum influence the occurrence of descent of this kind, that is to say, descent commencing in the vaginal wall? I think, in the first place, it has an indirect influence by often leading to incomplete involution of the vagina. If the perineum is torn and not afterwards united properly, the vagina is much less apt to involute completely than if the perineum was not torn at all, or if it was properly restored. Thus, by predisposing to subinvolution of the vagina and to laxity of the vaginal walls, the destruction of the perineum may be said to play a part in vagino-uterine descent. There is no doubt, also, as can be seen by inspecting the parts, that the perineum normally supports and lies against the lower part of the anterior vaginal wall. If the parts are inspected where the perineum is absent, the lower part of the anterior vaginal wall is seen exposed to view and has nothing to support it. The perineum acts as a kind of abutment, against which the lower part of the anterior vaginal wall normally rests, and thus offers it some natural support, and tends to prevent it being extruded. It may be said that the absence of the perineum does not make the vagina come down, and that it may remain in the normal situation when there is no perineum. This is perfectly true, and you will find in the poorer classes that it is comparatively rare for the perineum to be present in multiparæ. If it is true that failure to re-unite a ruptured perineum does promote subinvolution of the vagina, and if proper involution of the vagina does prevent this common form of descent, it is important either to prevent rupture of the perineum during childbirth, or if rupture does occur, to restore it as far as possible to its normal condition before the patient is left. Of those two things, the latter is much the more easy to fulfil. There is a common idea that the occurrence of rupture of the perineum is a slur on the skill of the practitioner, and the indirect result of that belief is that in many cases where the perineum is torn it is not sewn up again, so as to avoid exposing the supposed fault. But I think there has not yet been invented any device which will prevent the perineum rupturing in many cases. You will meet with people who will tell you—and I have met with many—that in the cases they attend the perineum never ruptures. That, on the face of it, is really absurd. The perineum ruptures in a certain proportion of cases of primiparæ. That it does not often rupture in multiparæ is due to the fact that the majority of these people do not possess a perineum, and therefore rupture does not occur for the best of reasons. Bearing in mind, then, that rupture of the perineum in primiparæ is frequently inevitable in normal delivery, there can be nothing to be ashamed of from the fact that it occurs. And if one does not boast of one's ability to prevent it there is no reason why one should not sew it up when it does occur. When people say they do this or that, and the consequence is that their patients do not have ruptured perineums, it implies that if there is rupture there must be some fault. As a matter of fact, whether the perineum ruptures or not depends infinitely more on the perineum than on the medical attendant. I do not, of course, mean to say that if forceps are used improperly you cannot undertake to rupture any perineum in primiparæ. But if the forceps are applied properly, and traction is exerted in the proper direction, in some cases rupture of the perineum can be prevented. A fact of some practical importance is this: that one of the most striking results of using axis traction forceps for the first time is to notice the extraordinary way in which the handles proceed to ascend over the woman's abdomen long before you think they should. That simply illustrates a fact which is often overlooked, namely, that the line of traction of the head is very much more forward than is commonly taught. The illustrations in certain text-books tend to perpetuate this error. As the child's head emerges over the perineum the handles of the forceps should look towards the patient's

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chin, with the patient lying in the ordinary obstetric attitude—on the left side, with the hips over the edge of the bed. The careful restoration of the perineum is of great importance, not only at the time but subsequently. At the time its restoration diminishes the risk of septic infection, and later on it leads to a more complete and perfect involution of the vagina, and thus to a more complete restoration of the condition of affairs which existed before childbirth. And it also lessens the risk of subsequent extrusion of the anterior vaginal wall in the form of a cystocele. The great point in sewing up a perineum is first of all to get the patient in a suitable position, so that you can really see the whole extent of the tear, and secondly to pass the sutures sufficiently deeply, so as to get at the apex of the tear, and not merely unite the skin. Uniting the skin only is of no value; the restoration of the perineal body is the important object in view.

With regard to vagino-uterine descent, there are degrees of this. The first degree is a condition of affairs where the anterior vaginal wall in the lower part extrudes when the patient strains, or in other words, when she increases her intra-abdominal pressure. And that can easily be seen if the patient is asked to bear down when the part is exposed. It is seen looking like half an orange, pink in colour, and becoming tense when the patient strains, because beneath the vaginal mucous membrane when extruded is a portion of bladder, containing more or less urine, which gives a sensation of elasticity to the protruding mass. This tense elastic swelling is called a cystocele. It is obvious enough that if the condition of affairs gets worse as time advances, more and more of the anterior vaginal wall will protrude, and the upper part of the vaginal wall being attached to the supra-vaginal cervix, it is clear that the cervix will be dragged down lower and lower, and ultimately the os externum and vaginal cervix will appear over the perineum. When any portion of the uterus shows externally the condition is spoken of as procidentia, which is merely a variety or degree of prolapsus. As long as the prolapse is limited to the vaginal walls, or as long as the uterus remains inside, the condition is spoken of simply as prolapse. In this way, then, the anterior vaginal wall is gradually extruded from below upwards, the lower part appearing first, then the middle, and then the upper part with the cervix attached; and as descent still further proceeds the posterior vaginal wall will become extruded from above downwards,—that is to say, the upper part will appear, then the middle, and then the lower part, though in the majority of cases the last inch never protrudes at all, being more firmly attached to the tissues behind than the rest of the vaginal wall. But when the upper three-quarters of the posterior wall are extruded, as well as the anterior vaginal wall and cervix, you have a mass covered with pink vagina, with an aperture near the centre which represents the os externum. In cases like this there are various anatomical points which are worthy of notice. In the first instance, as a result of traction, the os externum is usually gaping, and often admits the point of the finger. The vaginal cervix does not rise above the general surface of the vagina. When the patient strains, the tumour will become tense, owing to the urine being forced down into that pouch which accompanies the anterior vaginal wall. Posteriorly there is the prolongation of peritoneum known as Douglas's pouch, which extends down almost as far as the os externum. Into that pouch no intestine descends, except in those rare cases where the condition commences as a vaginal enterocele. If the mass is grasped in the hand from before backwards you will find, running up the centre what feels like a firm stalk about the thickness of one's finger, and this is the elongated supra-vaginal cervix. If a sound be passed through the os externum, it will be found to pass from four to five inches in the majority of cases, and this increase of the distance from the os externum to the top of the uterus is due entirely to that elongation of the supra-vaginal cervix, which has occurred as a result of this continued traction from the anterior vaginal wall. The sound will pass almost always with its concavity backwards, because the body of the uterus is lying in the hollow of the sacrum. In the vast majority of cases the uterine body remains in the pelvis, whilst the supra-vaginal cervix occupies the middle of the extruded mass. In certain cases the uterine body does come outside, and under those circumstances the fundus of the uterus will be found almost always to look directly backwards towards the anus. In cases of this kind the supra-vaginal cervix is not elongated, and the sound only passes two and half to three inches.

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I have only once observed a case where, although the uterus was still outside, it retained its anteverted and anteflected condition. That is due to the fact that in the majority of cases of cystocele the first thing that happens is the occurrence of backward displacement of the uterus. When the uterus looks forwards it obviously forms a distinct shield to the anterior vaginal wall, and cuts it off from that injurious pressure to which it is exposed after the uterus has fallen back. And there is no doubt that the removal of the protective influence I have mentioned is a first step towards the ultimate protrusion of that vaginal wall. You will now and then meet with a case of procidentia which has started in the reverse way to that described above. The lower part of the posterior vaginal wall has been extruded first, followed by the upper part, and then the cervix passes out beneath the pubic symphisis, and not over the perineum as in the former case. The descent still continuing, the anterior vaginal wall gradually descends from above downwards until both vaginal walls are completely extruded.

If the mass is replaced, and the patient is then directed to strain, it is usual to be able to see how the descent began. In the last-mentioned variety you will see the posterior vaginal wall lead the way, followed by the cervix, and then, lastly, the anterior vaginal wall will be everted from above downwards. I should here point out one difference between the early stages of extrusion of the anterior and posterior vaginal wall, that whereas in cases of cystocele there is almost from the first, if not from the very first, a certain amount of uterine descent, a rectocele may attain a considerable size without the neck of the womb being pulled down at all. It is only when the rectocele gets very bad that the uterus is dragged down. In some cases the rectocele may be as big as half an orange, and yet the cervix is found at its normal level. But sooner or later, as more of the posterior vaginal wall comes down, the cervix must follow it, and it passes out underneath the pubic symphisis, and the case is converted into one of procidentia. There is another point to be noticed about this mass, and that is, that on portions of its surface true ulceration may be seen. I say true ulceration because, as we shall see later on at another lecture, there is a condition of affairs which unfortunately is called ulceration of the neck of the womb, but which is not ulceration at all. The only true form of ulceration which is met with on the neck of the womb, except chancre or tubercular ulceration—both of which are exceedingly uncommon—is an ulceration which occurs in connection with procidentia. The ulceration is sometimes extensive, and generally occurs in the neighbourhood of the os externum, but sometimes it may involve the vaginal wall at some distance from the os. These ulcers are commonly known by the name of friction ulcers. The ulcers are shallow, and the surface is covered with a thin yellowish slough. They show no tendency to excavate or eat into the tissues, but spread at their margins, often in a serpiginous manner. They vary in size from a shilling to that of half-a-crown, and they secrete pus and give rise to a certain amount of bleeding, which is a matter of some importance. It is important because elderly women, if they are losing blood, will tell you of the loss of blood, but will generally omit to tell you that the womb has been hanging down between the legs for the last twenty years. Examination will make the source of the bleeding obvious. As a rule, the parts, even when ulcerated, are free from much tenderness, and can be handled readily without producing pain. No doubt the existence of these ulcers depends largely on the fact that the mass being constantly outside, the circulation through the part is interfered with, just as it is in a leg, the veins of which are varicose. In a leg, when the veins are varicose, the circulation of blood through it becomes impaired, and the vitality of the tissues becomes lessened. Hence superficial ulceration is apt to occur. In the case of the procident uterus the tissues are congested, the circulation through them is feeble, and thus nutrition is below the normal; a slight amount of friction causes superficial erosion, and sluggish ulcers form on the most dependent part. One of the best reasons for believing that this is the cause is that when this mass is replaced, and thus the interference with the circulation is checked, the ulcers usually soon heal; whereas, as long as the mass remains procident, you can never expect any healing to occur.

So much for that form of procidentia which results from the original protrusion of one or other vaginal walls, whether anterior or posterior; the result is the same whichever it is, and the procident mass is in an identical condition in both

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cases. I might point out here that the length of the supra-vaginal cervix will depend upon the resistance which the uterus has offered to the vaginal wall which has been dragging it down. You will remember that at the level of the os internum the utero-sacral ligaments are inserted, and that they run thence upwards and backwards towards the third bone of the sacrum. The anterior vaginal wall may be regarded as dragging on the cervix, whilst the utero-sacral ligaments hold it back, and it is obvious that the more firmly the utero-sacral ligaments hold back this part, the greater will be the elongation of the supra-vaginal cervix before this resistance is finally overcome. If these utero-sacral ligaments are very feeble, they will be able to offer but slight resistance to the downward movement, and the result will be that the supra-vaginal cervix would show but little elongation. In those cases where the utero-sacral ligaments, during the formation of a cystocele, are lax, the whole uterus may come outside, and may be felt lying in the mass which protrudes. Under such circumstances it will be found to be but slightly enlarged, and I think it is only in those cases where the utero-sacral ligaments offer but little resistance that the uterus finds its way out entirely into the procident mass. If there is great elongation of the supra-vaginal cervix, the whole uterus is too long to ever lie all outside.

Now let us consider the other condition of affairs, *utero-vaginal descent:* that is to say, where the uterus comes first. Of course, you produce utero-vaginal descent whenever you pull the os down to the vaginal orifice with a volsellum, although a certain amount of tensile elongation of the supra-vaginal cervix is also produced under these circumstances, as can be proved by the increased length which the sound enters. In this form of descent the uterus leads the way, and both the vaginal walls are inverted equally from above downwards. If you were to imagine the hand passed into the abdomen and the uterus thus to be thrust downwards, the condition of affairs met with in utero-vaginal descent would be produced. It is sometimes spoken of in English text-books as direct or *frank* prolapse, but the term utero-vaginal prolapse more correctly describes the condition. This form of prolapse may occur apart from childbirth. No doubt childbirth plays an important part in producing it, and no doubt it is more common in those who have borne children than in those who have not. The occurrence of pregnancy and labour often leads to relaxation of the utero-sacral ligaments, and if they do not undergo proper involution the uterus may fail in one of its natural supports. The uterus occupies the upper part of the vagina, and maintains its position partly, at any rate, owing to the tonicity of its own tissues. Thus if it is lax and flabby it may fall back, whereas, if it is firm it will stand up straight in the ordinary attitude. We may assume that many cases of backward displacement of the uterus are associated with a flabby condition of the uterus and a want of muscular tonicity. Utero-vaginal prolapse may occur in unmarried or sterile women, and it is remarkable sometimes at what an early age it may begin. It must be associated with some curious increase in the intra-abdominal pressure, and some peculiar decrease in the general tonicity of the parts which sustain the uterus. I have seen complete procidentia commencing in this way in a girl under sixteen, and I have seen it in a young woman of twenty-two or twenty-three, who stated that she had suffered in this way since the age of fourteen. Such a condition of affairs is unusual in the young unless they have borne children. Such occupations as washing or mangling predispose to this form of prolapse, just as they do to the vagino-uterine variety. Of course, this variety of descent, where the cervix protrudes outside and where more or less of the vaginal walls are inverted, can be easily recognised. The only thing it could be mistaken for in the first instance is a great elongation of the vaginal cervix. But it is the minor degrees of this descent which are, perhaps, a little more difficult to diagnose. A student who is just commencing the study of gynæcology frequently remarks that the cervix appears to him to be abnormally low down. The truth is it is lower than he expected to find it, for after all, the distance of the cervix from the vaginal orifice is not very great, perhaps two and a half or three inches, and that represents the normal distance of the os externum from the orifice of the vagina. Thus one is led to believe that there is some descent, especially if the patient complains of bearing-down pains. Now, the cervix will appear abnormally near the vaginal orifice if the vagina is unduly short. Shortness of the vagina

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is not uncommon, and if that is so, the cervix may be said to be unduly low down, and yet there may be no prolapse. If the vagina is short the os will be found at a comparatively small distance from the hymeneal orifice. Another condition which may lead to the cervix appearing nearer to the vaginal orifice than usual is undue length (hypertrophy) of this structure. This condition is generally congenital. Another cause, of course, is actual descent or prolapse of the uterus. If you want to find out whether the uterus is lower than it should be, that is to say, whether there is some utero-vaginal prolapse or not you must remember the two things I have mentioned which may simulate it, and ask yourself whether the vagina is unduly short or the cervix unduly long. If the cervix is low down because the uterus is prolapsed, it is easy to push it up. If you cannot push it back you may be sure that it has not descended. If it is near, and it has really descended, you will be able to push upon it, and it will then retreat, and it returns again as soon as the patient stands up or strains. In that way you can readily determine whether a condition of descent is actually present. In the other two cases, pressure does not cause retreat of the uterus. Another very curious thing is that there is nothing so misleading, at any rate to the beginner, as an exploration of the pelvic organs through the rectum. One of the things which strikes one, and it is a constant source of surprise, is that the cervix appears to reach extremely close to the anal orifice, the observer probably having had the impression that it was three or four inches up. Remember that the cervix is felt easily by the finger in the rectum, and seems to project into the anterior rectal wall. Direct descent will lead, in time, to the appearance of the cervix at the vaginal orifice, the vaginal walls being inverted. In such cases the uterus is not found to be elongated, the sound only passing two and a half or three inches because the supra-vaginal cervix has not been elongated. The uterus has been pushed out, and not pulled out.

Now I want to say something about treatment of this form of descent. You will, of course, understand that to-day I am not speaking about the minor forms of displacement. Remember, one cannot speak of the treatment of these cases indiscriminately. We will consider those cases of descent or protrusion of either the anterior or the posterior vaginal wall, in other words, the treatment in the first instance of either cystocele or rectocele, because in the majority of cases one sees these cases at that stage. It is well to remember that if the cystocele has once well developed there is no form of operative treatment which can be said to cure it. Cases of extensive procidentia are often sent up for operation, but in many instances they are quite unsuitable for anything of the kind. However, it is important to remember that, as far as cystocele is concerned, there is no plastic operation about the vaginal wall which will cure it. The removal of a portion of the anterior vaginal wall will not restore the patient to the condition she was in before. In cases of cystocele generally all that can be done is to give relief by means of some sort of support. You may cut all kinds of figures in the anterior vaginal wall and bring the raw surfaces together, but in a few months the whole thing will be as bad as ever it was. If the perineum is restored it might keep up the anterior vaginal wall if it were in a normal condition, but it will not keep back one which has become stretched and lax. When a cystocele exists we are practically reduced to supporting it by some form of instrument. There is no doubt that on the whole a watch spring or rubber ring is the best form to use. If it occurs shortly after childbirth or within a few weeks of it, at a time when the woman is still weakly and out of health, when we may hope for still further involution of the parts, or when the woman is suffering from oversuckling, a good deal of improvement may follow attention to the patient's health, and the use of astringents locally. But when the condition has been in existence for some months, much improvement cannot be looked for, and it can only be dealt with by wearing a support. When patients ask you how long they are to wear the support you have to say that there is not much prospect of their being able to leave it off. I mean patients with distinct cystocele, not merely a slight bulging of the anterior vaginal wall. A well-marked cystocele means a permanent wearing of some kind of apparatus. I am sure it is quite a mistake to offer her any hopes of cure from any operation. It is desirable sometimes that the perineum should be restored; but very often there is no object in restoring it, provided that the vaginal orifice is

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narrow enough to enable the ring to be readily retained.

I have spoken of cystocele and rectocele. A cystocele is well held up by a ring, the upper part of the ring being behind the cervix, the lower part three quarters of an inch from the vaginal orifice. Rectocele, on the other hand, very often is not kept up by a ring at all. In some cases the lateral stretching of the vaginal walls is sufficient to prevent extrusion. Rectocele, unlike cystocele, is, comparatively speaking, amenable to plastic operation. A rectocele of considerable size does not occur when there is a perineal body, and if this is restored and the superabundant vaginal mucous membrane excised, it very often does away with rectocele, and enables the patient to get about without wearing a support at all. Cystocele does not lend itself to any form of operative treatment; rectocele, on the other hand, does. Cystocele is, fortunately, always kept up by a simple form of instrument, whereas, rectocele is not.

Let us consider what are the means of dealing with the cases where a descent is of a more extensive scale, where procidentia is marked, where there is a cocoanut-shaped mass protruding. There is no difficulty about reducing it. Sometimes the parts are tender, but there is no real difficulty in reduction of such a procident mass. If the cervix and the vagina have become dry and cracked, anointing the parts with vaseline will facilitate their return. In such a case the uterus, instead of measuring two and a half inches, measures four or five inches. The supra-vaginal cervix is long, and the body not appreciably altered in size. On making a vaginal examination after replacing the mass, the body of the uterus is generally found looking backwards in the hollow of the sacrum, and the long neck makes it difficult to get the body up above the pelvic brim. In some of these cases a ring is sufficient, and, especially in elderly women, when a ring can be retained it is by far the best method of treatment. Sometimes the vaginal orifice gapes to such an extent that no ring can be retained, and in a young woman if that is the case one may be tempted to see what operative treatment will do. Before introducing a ring in these cases it is always desirable that the body of the uterus should be pushed up as much as possible, so that the upper margin of the ring should not rest against it. If the ring is not retained two courses are open to you, either to suggest to the patient that she should wear some form of instrument which is retained in the body by means of tapes and a belt, or to undergo some form of operative treatment. There is a third alternative, and that is to introduce into the patient's vagina one of those numerous pessaries which may be classed under the head of shelf pessaries. A shelf pessary is one with a disc-like top, and a stem coming down from the lower surface. They are always made of hard material, sometimes wood, sometimes vulcanite; sometimes the shelf is circular, sometimes oval; sometimes the shelf is made to shut up so as to render its introduction more easy; but they all act like a shelf, and the edges press firmly against the vaginal walls. The stem, which comes off from below, prevents the shelf from turning round and coming out edgeways. It must never be forgotten that this form of pessary will inevitably cause ulceration of the vaginal walls if retained for any length of time, and that a vesico-vaginal, or recto-vaginal fistula will result. Such a pessary, if employed, should be removed every night by the patient, and replaced in the morning. If she is unable to do this she should not be allowed to wear it.

Just a few words with regard to those pessaries which have a stem which comes down, and protrudes externally, and are kept in by means of tapes attached to a belt around the waist. Such a form of instrument must be inconvenient, to say the least of it, and although it is the best that can be done for certain patients, it is not to be advised, at any rate in the case of those who are not too old to undergo any operation. It is true that all operations for procidentia are more or less unsatisfactory, but still, in a certain number of cases, by doing two things—by removing a considerable portion of the greatly elongated supra-vaginal cervix, and by, at the same time, restoring the perineum—one may enable the patient to keep her womb inside her body by means of a simple ring. A ring never causes serious injury, though perhaps it causes a little superficial ulceration, or vaginitis; but it never ulcerates through into the surrounding viscera. I have seen rings which have been *in situ* for many years without causing serious injury, although causing an offensive discharge from the vagina.

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The only other point I will allude to is, that many people imagine that in cases of this kind it is possible to keep up the womb by more extensive operations, such as abdominal fixation. Of course, if you attach the womb, in the case of procidentia, to the anterior abdominal wall, as you can easily do by making an opening in the middle line in front, and stitching it to the anterior abdominal wall, it can be kept there for a short time. But directly the patient walks about the intra-abdominal pressure will bring it down, and the adhesion formed will gradually stretch until the whole mass protrudes from the vulva as before. Abdominal fixation may be taken to be out of the question in cases, at any rate, of procidentia commencing as a cystocele or rectocele, and shortening of the round ligaments is equally inefficacious.

In cases of direct or utero-vaginal descent it is possible that abdominal fixation might give better results.

In most cases, however, the chief reliance is placed on the ring pessary.

Infrequent Cases of Migraine.—Pässler 'Münch. med. Woch.,' July 1st, reports three cases of migraine in illustration of features of this disease, which are of unusual occurrence. The chief interest concerning the first case relates to its etiology. The patient was a perfectly healthy young man, who first developed attacks of migraine after having received a blow upon his head. The attacks at first were somewhat indefinite in nature, but soon became typical in character. Hitherto very few cases have been reported in which the origin of the disease could be directly traced to traumatism. The second case showed, during some of the attacks, vaso-motor disturbances, a bilateral dilatation of the pupils, and a non-reaction of the pupils to light. It is interesting to note that in those attacks in which the vaso-motor disturbances failed the eye symptoms were also absent. The third case was characterised by disturbances of motion which closely resembled those seen in cerebellar ataxia; the tendon reflexes were also markedly increased. The principal treatment in this case was quinine, from the administration of which some benefit was produced.—*Medical Record*, July 26th, 1902.

A CLINICAL LECTURE

ON TWO CASES OF

VENOUS THROMBOSIS IN HEART DISEASE.

Delivered at Guy's Hospital, May 28th, 1902.

By W. HALE WHITE, M.D., F.R.C.P.

GENTLEMEN,—To-day I want to tell you chiefly about a rare complication of heart disease of which there is at present an example in the hospital. The woman has been in so long that probably many of you will remember her. She was in No. 14 bed, but is now in No. 17 in Mary Ward. Her name is Harriet H—, her age is 24, and she came in so long ago as the 25th of last September. She said her father had had rheumatic fever, and she herself had had it previously. With regard to the present illness, she told us that she had been ailing for seven weeks, when she had a sore throat. You will notice there the importance of sore throat as one of the manifestations of the rheumatic state. Since the sore throat she had had pain first in one joint and then in another. And there you notice also the important factor in rheumatic fever of the pain flying about from joint to joint. At present she complains of the pain in the elbows and knees, and she is breathless on exertion. That, of course, suggests that she has some endocarditis, as she has rheumatic fever. The full report of the case is very long, and I have abstracted it for you. On admission the temperature was 100·4°, and the patient was sweating profusely. You will notice that there is the sweating in connection with rheumatic fever. The history also illustrates the recurrent character of rheumatic fever, because she had had it before. Notice also that it is a family disease, for her father had it. Then, the cardiac impulse was diffuse and heaving, and the cardiac dulness extended a little outside the nipple and a little to the right of the sternum. There was a loud systolic murmur audible at the apex, traceable round to the axilla. You could also hear at the apex a presystolic murmur and an early diastolic murmur. Both these were limited to the apex. Over the aortic area at the second right costal cartilage you could hear a loud to-and-fro murmur indicative of aortic obstruction and aortic regurgi-

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tation. So you see the diagnosis was comparatively easy, namely, that the patient was suffering from rheumatic fever; that she had endocarditis of her mitral valve, leading to mitral constriction and regurgitation; and endocarditis of her aortic valve, leading to aortic obstruction and regurgitation. Now, it is unusual in rheumatic fever to get all four valves affected in that way. And you should remember that it is almost always the mitral valve that is affected first, and there is every reason to believe that in this case the affection of the aortic valve took place secondarily to that of the mitral. She was treated in the usual way; that is to say, she was given considerable doses of salicylate of sodium, ten to twenty grains every three hours to begin with, and she was kept absolutely at rest in bed, and put upon a milk diet. Rheumatic fever is such a common disease that you all know the treatment of it. Many of us, in fact I think most doctors, believe that in salicylates we have a genuine specific for rheumatic fever, not perhaps quite on the same footing as mercury, which is a specific for syphilis, for even after cure by salicylates the disease may recur. We know that large doses must be given. Under this treatment the patient improves, the temperature comes down; and we know that it is equally important to keep the patients upon a milk diet, and I am always pointing out to you in the wards that if you want to prevent relapse you must keep up the diet of milk for ten days or a fortnight after the temperature has become normal and the pain gone. On this treatment our patient seemed to be doing well until November 2nd, and then she again illustrated how rheumatic fever is a recurrent disease, for she had another attack of it. It implicated many joints, her temperature went up, and on November 4th we were more anxious about her because she complained of much shortness of breath and cardiac distress. On November 8th there was a loud pleuro-pericardial rub to be heard; that means a rub due to the friction of the lung against the pericardium. In this case it was more dangerous than a pleuritic rub elsewhere, because it suggested that there might be, or soon would be, pericarditis. You will notice that that is a common complication of rheumatic fever. She was complaining of precordial pain which came and went irregularly, and on November 11th the cardiac dulness, which had lessened since admission, had again increased, and now extended nearly an inch to the right of the sternum and outside the left nipple line. That, again, suggests to you that when people get rheumatic fever they also get myocarditis, or an inflammation of their cardiac muscle, with the result that the heart is particularly prone to dilate; and, as I was saying in the ward only yesterday when speaking upon the same subject, there are probably many hundreds of people going about at the present time with cardiac disease that might not have been as bad as it is if it had been remembered—or if the patients had obeyed the doctor—that it was necessary to keep in bed for a very long time when they had their rheumatic fever. You must, by keeping the patients in bed, rest the heart and prevent the dilatation which will otherwise follow; that is to say, if you get them up. If you believe from the dilatation or from the murmurs that the heart itself is affected in rheumatic fever, it ought to be weeks, often as long as six weeks, before you get the patient up, because by resting him at that stage you may prevent his becoming a case of permanent cardiac disease. On November 13th a well-marked pericardial rub was heard, and that, of course, shows you how common pericarditis is as a complication of rheumatic fever. The patient, who had been taking digitalis, had been troubled with vomiting, and so two drachms of the infusion of digitalis were given *per rectum* every two hours, and the rectum was washed out daily with a normal saline solution. The cardiac region was at this time very painful: aortic and mitral murmurs could be heard, and also pericardial and pleuro-pericardial rubs. The patient was in considerable distress; the cardiac dulness in the position of the apex remained unaltered, and she was ordered ℥xv of nepenthe, to be taken three times a day in water, and was put upon small quantities of milk every two hours, and three leeches were applied over the pericardium. We will come back presently to the treatment of pericarditis. On November 20th we noticed systolic retraction of the cardiac apex. This is often correctly interpreted as a sign that the apex of the heart is adherent to the parietal pericardium, so that when the heart contracts in systole the pericardium is dragged in, and it drags in the chest-wall with it. It is a sign of importance, but its importance is much exaggerated,

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because a pericardium can be adherent without there being a systolic retraction of the apex; and you may see something there like it in people in whom there is no reason to suspect adherent pericardium. But if the patient has had pericarditis and then the apex is retracted during systole, it is very strong evidence that the pericardium has become adherent to the heart. From this date to December 1st the patient's condition may be summed up by saying she was very ill; her temperature was raised usually about two degrees, her pulse was rapid, often weak and irregular; the cardiac apex remained in the same place, and the pericardial rub was sometimes present, sometimes not. The pleuro-pericardial rub was usually audible. The patient was kept absolutely on her back, and she was given enough morphia to keep her gently asleep. Digitalis was given by the mouth or by the rectum. If there was nausea, it was given only in small quantities by the mouth, or only by the rectum. She took a little brandy.

And now I come to the features which made this case the subject of a lecture. On December 1st some œdema was noticed on the left shoulder and upper arm, and it was present to a less degree on the left side of the neck. She was very short of breath, and somewhat livid about the face. Oxygen inhalations were used, and we discussed the question whether the œdema was due to thrombosis or to mediastinitis. The patient had been so ill that her back had not been examined for many days; so to-day we rolled her on her side, and extensive signs of pleurisy were found on both sides, with signs of fluid at the base of the left lung. The probable presence of thrombosis made me suspect that the fluid in the chest might be pus, so an exploring needle was inserted into the chest on the left side, but only clear fluid was withdrawn. The fluid was submitted to Mr. Pakes, who reported that it was sterile. Towards evening the patient became very weak, and it was necessary to inject strychnine subcutaneously. On December 12th the œdema on the neck and arm was much more marked, especially on the left side, and it extended down to the fingers and also a little over the upper three intercostal spaces on the front of the chest. On pressing over the right shoulder and the right clavicle and the side of the neck, there was considerable tenderness; the patient did also complain a little of pain in the neck, but it must be remembered that she was often having morphia. The face was cyanosed. From this time the œdema gradually got less. In a few days it was possible to feel both external jugular veins; they were hard, as thrombosed veins are. On December 20th and 23rd she had two severe attacks of dyspnœa, with lividity, and on each occasion that was relieved by oxygen and by venesection. From this time there was continuous but very slow improvement in all respects. She often had severe dyspnœa on the slightest exertion, and that was not surprising when we remember that she had aortic obstruction and regurgitation at her mitral valve, and also an adherent pericardium.

Now I will refer to my second case, and then I will tell you about the facts of the cases. The next patient is a man æt. 26, whom I saw on April 6th of this year, with Dr. Daunt. The patient had been previously under his doctor's care eight weeks ago for influenza, and before that was well he had insisted on going to Devonshire strongly against his doctor's advice, when he was too weak to undertake the journey. His doctor in London has been told that while there he had pneumonia and was very ill. He came home a month ago, when he was still far from well; he insisted on going to his work, so there was no proper opportunity of examining him. For two or three weeks he probably has had dullness at the base of both lungs. On April 5th he began to get livid about the head, face, and neck. When I saw him on April 6th the head, neck, and to a less degree the upper two inches of the front of the thorax, the arms, and the finger-nails were all livid. The rest of the body was of a healthy colour. The veins on the chest were marked, and also those on the neck. Both external jugulars felt hard. The livid part was somewhat œdematous, that is to say, both arms, the face, and the neck. The pulse was rapid and weak; both pulses were equally weak. The heart appeared a little dilated, the apex being outside the nipple line. The heart-sounds were very feeble, and I thought I detected a faint systolic murmur over about the position of the middle of the heart. Still I was not sure about it. I thought there was some little dulness in the second right intercostal space, and that suggested to me the possibility of an aneurysm, but I soon dismissed it from my mind. The patient was very young for such a condition; and aneu-

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rysms by pressing upon veins do not, as a rule, produce lividity. As a rule, in order to produce lividity it is necessary to block the inside of the vein. Therefore, if the case is one of either aneurysm or growth, and the veins are much dilated, it is very strong evidence that of the two the diagnosis of growth is correct, because the growth will grow into the interior of the vein and thrombose it, while aneurysms will just stretch it. Now, to go back to this patient; the air entry into both lungs was equal. The history was very imperfect and difficult to get; but bearing in mind that he had had pneumonia, and that the bases of both lungs were very dull and he was very livid, I thought he possibly might have an empyema, for this makes people very livid. I did not think it likely, but to be sure I put the needle into both sides of the chest, but in neither case was any fluid withdrawn. On the whole, therefore, the diagnosis made was that it was most likely the patient suffered from influenza, afterwards with pneumonia, and had some thrombosis of the veins opening into the superior vena cava. He was so livid that we wished to venesect him, but he and his friends objected, and therefore three leeches were applied over each side of the neck, and the leech-bites were fomented so that the patient bled considerably. On April 8th I saw him again, and then I found that he was much better. The previous œdema about the arms, the neck, and upper part of the chest was less, the veins on the thorax were still well marked, and there was a little ring of dilated veins about the lower part of the chest. There was no doubt now about the systolic murmur; it was loud and audible at the apex of the heart, and was conducted to the axilla. There was a well-marked cardiac impulse in the sixth space, half an inch outside the nipple line. I thought the probable reason that the previous cardiac impulse had been so diffuse and the murmur had not been heard was this: that owing to the great venous distension in the neck the right heart was dilated, and the whole heart could not contract properly, and so there was not enough force in it to make a murmur.

So in both these cases we have patients who had lividity and œdema of the arms and head and neck. When people have lividity and œdema of the arms and head and neck it is quite clear that there must be some block in the flow of blood back through the superior vena cava. What we have to do today is to think what that might be.

As I mentioned just now, both growth and aneurysm should come into your minds as possibilities. But both these patients were suffering from heart disease, and we wanted their œdema and lividity to fit in with their heart disease. Further, there was no evidence in either patient of growth or of aneurysm. Both the patients were young for such conditions, and we were able, by going over the cases, to quickly put these out of court. The two other things which were suggested at the bedside were that there was some mediastinitis, or that there was some thrombosis of the face at the root of the neck. Fortunately it so chanced that in the course of medical reading while the woman was in the hospital I happened to read an account of a case of heart disease with thrombosis of the veins of the upper part of the body, recorded by a Dr. Ford, of Montreal, in a Canadian medical journal. That gave me the reference to some cases which have been recorded by Professor Welch, who read his paper before the Association of American Physicians. And our two cases fit in so well with his description of that rare complication of heart disease, namely, thrombosis of big veins opening into the superior vena cava, that I thought we might profitably, this afternoon, compare our two cases with the description he gives of that rare condition. You know thrombosis, as I have often pointed out to you in the wards, is a common disease. The two acute diseases which frequently lead to it are typhoid fever and influenza; and the two chronic diseases which lead to thrombosis are tubercle and cancer. Now I mention that because it is very important you should remember that so far from thrombosis of the systemic vessels being very common in heart disease because of the difficulty of circulation, you see that it is not so, it is a very rare complication, and I have not myself seen it before this winter. Diseases in which there is probably no particular difficulty of circulation, namely, typhoid fever, influenza, phthisis, and cancer, are those diseases in which over every other thrombosis is frequent. That reminds us of what one is always pointing out, that the clotting of blood inside the body is not due to any single factor as far as we at present know. At any rate that single factor is not the retardation of the circulation. When I say

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that thrombosis is very rare in heart disease, please remember I am strictly confining my remarks to the general systemic veins. You know thrombosis is very common during heart disease in the branches of the pulmonary artery, in association with the ordinary pulmonary apoplexies which you so frequently see in the lung. And then, also, thrombosis in the arteries in heart disease is not uncommon in those cases where there is atheromatous change of the aortic valves. You get atheromatous change in the arteries of the body, and thrombosis in the atheromatous vessels.

Now there comes this fundamental fact about this rare condition. The thrombosis of the systemic veins that occurs in heart disease occurs nearly always, as our two cases illustrated, in veins of the upper extremities and the head and neck. It is difficult to know the exact proportion, because the cases are so few; but it is something like fifty to one in favour of the arms and head and neck rather than the other veins in the body. That is a remarkable thing, and shows that it is a special complication of heart disease, because you will remember that in influenza and typhoid fever and tubercle and cancer—the other four diseases that frequently lead to thrombosis—the thrombosis is nearly always in the veins of the lower extremities. So in nearly all common cases of venous thrombosis it is the veins of the lower extremities which are affected, but in heart disease, for some reason which we do not know, it is the veins of the upper extremities and of the head and neck. Now, of twenty-two cases in which Professor Welch was able to get the age and sex of the patients he found seventeen were females and five males. That is probably, if anything, a higher proportion of females as against males than the fact I will point out to you directly, namely, that this complication of heart disease is much more frequently associated with mitral than with aortic trouble, should warrant. With regard to age, our cases illustrated the point that this form of thrombosis occurs in young people between fifteen and thirty years of age. The woman was twenty-four and the man twenty-six. In every case but one of Dr. Welch's series there was mitral disease, organic disease of the mitral valves. Both our patients confirm that, for they both had organic disease of the mitral valves. And sometimes the aortic valves are affected too—as a matter of fact, ten times out of the twenty-two. Our cases bear that out also, because one of our two had the aortic valves affected also. There was in Professor Welch's series always mitral regurgitation, and there were often symptoms of mitral obstruction too. And that harmonises with our experience, for both our patients had mitral regurgitation, and one of our patients had mitral obstruction as well. In all the recorded cases the valvular disease was chronic, and in one quarter of them acute conditions supervened upon the chronic. That likewise harmonises with our experience, because both our cases were clearly instances of chronic heart disease, and in the woman it is probable that some acute trouble supervened upon it. In one quarter of Professor Welch's cases there was adherent pericardium. Our cases remind us of that fact, because the woman had an adherent pericardium. So we may sum up by saying that this form of thrombosis is particularly found in young adults suffering from mitral disease, and that to some extent explains that it is much commoner in women than in men, because mitral disease is commoner in women. Another point is that it appears during the period of extreme failure of the heart. Many of you here have seen our female patient, and you will admit that if any one was in a state of extreme failure of heart that woman was. And that was true also of the man. From this it follows that there is often tricuspid regurgitation and other symptoms of serious heart disease.

Now it is important you should know the locality of the thrombi, and I will put up the figures in regard to that on the blackboard. Out of twenty-four cases the thrombosis was on the left side in twenty-two; and of those twenty-two fifteen were unilateral and seven bilateral; that refers to unilateral as regards the arm and one side of the neck. It was right-sided only in two. So you see there is the curious clinical fact which both our cases illustrated, that the left side is very much more often affected than the right. But our cases do not quite accord with the above distribution, because in both our cases the thrombosis was bilateral, and it is more frequent to get it unilateral than bilateral. The rarest thing is to get the veins on the right side affected, and not the left. In seven of the twenty-two cases it was limited to the arm, in one case it was limited to the neck, and in sixteen it was in both. That corresponds exactly with our experience, because in both our

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cases both arm and neck were involved. Inasmuch as the thrombosis is frequently bilateral, you get both internal jugulars, both the subclavians, and both innominate veins affected, and you will remember that in our cases we felt both external jugulars to be involved. The brachial and axillary veins are also affected, and that is about the order in which the veins are found to be thrombosed. In very severe cases even the superior vena cava itself is thrombosed. Post-mortem experience shows that the starting-point of the clot is at the lower part of some vein, for instance, the internal jugular, at the lower part of the innominate veins where they join, or at the lower part of the subclavian or external jugular, as the case may be. Also, post-mortem experience seems to show that as a rule the walls of the veins themselves are not diseased, as well as the fact that very often the lymphatic glands in the neighbourhood of the veins are actually inflamed.

I have pointed out to you that this condition exists, and I do not think the symptoms need detain us long. There is, of course, pain. The woman complained of pain over the affected area. All thrombosed veins are tender. There are also lividity and œdema over the affected area. Then, when the œdema has gone down and you come to feel the veins, you will find that they are like hard thrombosed cords. There is only one point about the œdema to which it is necessary to draw your attention particularly, and it is this, that the œdema in any severe case usually extends about two inches on to the thoracic wall. So we might sum up the symptoms of this disease rapidly as a quickly appearing lividity and œdema affecting the face, neck, and upper extremities, and extending two inches on to the thoracic wall; that the patient complains of pain and tenderness; and that the course of the veins can be felt. Those are really all the symptoms except that there is also often distension of the superficial veins. You might say that thrombi in other parts of the body usually cause symptoms, such as rigor and fever, but any general symptoms due to thrombosis in heart disease are usually over-shadowed by the serious illness of the patient due to the heart disease. Bacteriological examination has only been made in three fatal cases. In two it was negative, and in the remaining case *Streptococcus pyogenes* was found in the clot. As you would expect, it is a very serious symptom, and out of twenty-four cases twenty-two died. The wonder is that the woman upstairs did not die; indeed, neither she nor the man are yet out of the wood. So we cannot say with regard to our two cases whether there will be a fatal or a successful termination. I think the woman will recover, but the man is still very ill.

I cannot do much, I am afraid, in explaining this peculiar localisation of thrombosis to you. It is quite different to the localisation of venous thrombosis in other diseases, but it has been suggested that the greater length and obliquity of the left innominate vein accounts for it occurring largely on the left side. It has been suggested that the position of the valves in the left internal jugular, external jugular, and subclavian veins would favour clotting. Whether that is so or not I do not know. It has been suggested that in these cases the dilated left auricle will press upon the left innominate vein, and that some of the patients, like the woman, may have considerable pleuritic effusion, and that the fluid will press upon the innominate vein. But after all these are only suggestions, and I give them to you for what they are worth. Professor Welch himself is inclined to think that thrombosis generally may be related to a kind of whirling movement of the blood. He says he thinks that in these cases you are very likely to get such whirling movement. He suggests that the cervical fascia holding the veins open, that the disposition of the valves, that the bulbous end of the internal jugular, that the meeting of currents of blood at different angles, that the backward flow of blood because of the regurgitation through the tricuspid, will all help it. But after all, as I say, these are merely suggestions. He thinks the determining factor is really the presence of bacteria. That is all I have to tell you about this rare complication, and I brought the cases to your attention to-day because venous thrombosis is a rare complication of heart disease which has not yet been often described, and therefore this discussion may be useful.

The other point I wanted to refer to is the occurrence of mediastinitis. This is very rare, that I am sure of; the word means inflammation of the areolar tissue of the mediastinum. In ten out of eleven cases the inflammation is suppurative, and is frequently due to a blow or to caseous glands suppurating. In neither of our cases was there

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any evidence of pus. Those cases form a category by themselves. We have now left a rare mediastinitis which is not suppurative, and that is what is commonly called chronic mediastinitis. That is an excessively rare condition; and it is only, I think, one or two years ago that Dr. Frederick Taylor gave an address on the subject. I have nothing more to tell you in addition to that. Chronic mediastinitis is very frequently associated with pleurisy and with adherent pericardium—if you can call anything frequent in such a rare condition,—therefore adhesions of the pleura and adhesions of the pericardium quite overshadow the inflammation of the cellular tissue of the mediastinum, and make the diagnosis of mediastinitis at the bedside very doubtful. One point which does help you with the diagnosis is, that these rare cases very frequently have some chronic peritonitis as well. So we might sum up the condition of chronic mediastinitis in what I have told you. We may say it is a very rare condition indeed, associated particularly with adherent pleura and adherent pericardium and with chronic peritonitis, and that during life so many of the severe symptoms are due to adhesions of the pericardium and adhesions of pleura, and possibly also to chronic peritonitis, that it is almost impossible to disentangle the symptoms so as to assign correctly which symptoms are due to mediastinitis and which are due to the other conditions. But we did suggest at the bedside that the woman might have this condition, because you see it accounted for a sudden increased pressure on the superior vena cava and its branches, and she had an adherent pericardium and adherent pleura, and we knew that mediastinitis was associated with these conditions. I need hardly tell you that the presence of the thrombosed veins at once put this out of court, and at the best it was not a very good suggestion, for the difficulty of it was this, that her œdema and her lividity were confined to the upper part of the body, so we should have to suppose that her mediastinitis, while it did still hamper considerably the return through the superior vena cava, was not troubling her much as regards the return to the inferior vena cava. And there is so little vena cava in the thorax that that might have been so. But as a rule people with mediastinitis suffer from general œdema and lividity.

And the last point I wanted to draw your attention to is, as to what you are to do with a patient who has acute pericarditis. The woman's case illustrated the points so admirably that I cannot resist the temptation to lay them before you. By attending to these points properly you not only save the patient's life, but you may determine whether she shall be a perpetual chronic invalid, or whether she shall be in a position of fairly good health. The adhesion of pericardium to the heart does not appear to matter very much, provided that the adhesion takes place when the heart is of normal size; but if the adhesion takes place when the heart is dilated, the adhesions will always hold the heart stretched out; and then no amount of treatment can do the patient much good. So your object is to treat the patient in such a way as to prevent the heart getting dilated, but to obtain adhesions upon the heart in its normal size, and not when it is a great flabby organ. The reason why the heart dilates so readily is that there was never a pericarditis without some co-existing myocarditis, often in considerable degree, and also the fact that the pericardium itself becomes soft and sodden, so that it easily gives way. Now that I have told you that, you all know what to do. Your object is clearly to prevent in every possible way the heart from dilating; and the best way to do that is to give it very little work to do, so as not to increase the pressure within. So first of all keep the patient absolutely at rest in bed. We did not sit our woman patient up for many days together to examine her back, and I am sure we did right. What could we do if she had a pleuritic rub? We could have done nothing which would have compensated for the harm that might have followed in the shape of dyspnœa and cardiac dilatation. So be sure to keep your patient absolutely quietly at rest in bed.

We will take the question of diet next. What have we to aim at? You want to prevent dyspepsia, because such a patient, on account of the condition of the mucous membrane of her stomach, is very likely to have dyspepsia. There are the dilated veins in the stomach, and there is a coating of mucus, and in view of these facts you must feed your patient accordingly. What could be worse than for such a woman as our patient to get flatulence and dyspepsia? The flatulence pushes up the heart and makes its action irregular. Therefore, at all costs, prevent flatulent dyspepsia. We

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kept her upon small quantities of milk and easily digestible food, giving her only very small quantities at a time and at frequent intervals, because we did not want to distend the stomach. And for the same reason, because we did not want her to be dyspeptic, we kept the bowels regularly acting, and rather than have vomiting and flatulence you must feed *per rectum*.

The key-note of the next step in the treatment is again to rest the heart as much as possible. During sleep our heart's action is some ten beats slower per minute than when we are awake. You will see what an enormous amount of rest is obtained for it if you multiply ten beats per minute by sixty, and then that by the number of hours sleep. So we kept our patient comfortable and quietly drowsy. As a rule she did not notice us very much when we came into the ward; she slept very well at night, and she slept in the afternoon, because we gave her nepenthe. If it will not be absorbed by the stomach you must give opium by subcutaneous injection; it is most valuable in the treatment of pericarditis.

The next point is one in regard to which I think you will find people are often, I believe, in error, and that is in regard to the administration of digitalis. They say you should not give a patient with severe pericarditis digitalis, because it increases the amount of work which the heart has to do. That may be so, but you slow a heart which is usually rapid, and you slow it by prolonging the diastole. It is an ideal drug. A heart is perhaps beating at 120 per minute, and you bring its rate down to 80. In that case, therefore, you slow its beats 40 per minute by prolonging the diastole, and this rests the heart. Just look at the increased length of diastole every minute. Another thing is, digitalis tones up the heart generally and contracts it, thereby helping us to get adhesions on a contracted instead of on a dilated organ. So I am sure I have often seen good effects at the bedside by the prolonged rest which digitalis gives to the heart, and by the fact that it tones it up and keeps it contracted. The advantages of digitalis in pericarditis greatly outweigh the disadvantages. I have not infrequently seen patients who were very ill with pericarditis immensely improved directly digitalis was given. But it is a drug which frequently produces vomiting; therefore, the moment there is any suspicion of vomiting, you must give the drug *per rectum*. Digitalis was given *per rectum* in our patient.

Again, I do think that sometimes—though I cannot explain why—you do the patients good by putting some leeches over the heart region; at any rate, there is no doubt that the leeches relieve the pain. I think those are the chief points to which I wanted to draw your attention in treating pericarditis.

Serum in Scarlet Fever.—Since the discovery of antidiphtheritic serum a number of experiments have been attempted, often with little success, to apply the same principle of progressive immunisation to various animals by means of cultures or toxins of microbic origin, with the view of obtaining curative serums for other infectious maladies, such as cholera, smallpox, typhoid fever, etc. The eruptive fevers, and in particular scarlet fever, have not lent themselves to these researches, because the agents producing them are not very fully known; but, as a first attempt to answer the general question of immunisation in these allied diseases, it has been asked whether the serum from the blood of subjects recently cured of scarlet fever, for example, might not be capable of exercising favourable influence against this disease? The writer has shown that such injections seem to make the patients feel better, but do not have a very definite effect upon the various other symptoms, such as the fever and local signs. The results were, in short, somewhat contradictory. E. von Leyden, O. Huber, and F. Blumenthal have also carried out a similar series of experiments. Von Leyden injected only ten cubic centimetres of serum from convalescents, and later twenty cubic centimetres without even seeing bad symptoms. He estimates, furthermore, that with advantage this dose may be increased. It goes without saying that it is of the highest importance to be certain that the subject furnishing the serum is exempt from various diseases. This serum-therapy was applied in about sixteen cases. One of them was a woman who, after an abortion, suffered also from scarlet fever. In her case the result was remarkable. In fifteen other patients, six gave positive and nine doubtful results after treatment. Von Leyden thinks that his experiments have proved great possibilities from this form of treatment.—Weisbecker, *Monthly Cyclopædia*, July, 1902.

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A CLINICAL LECTURE

ON

SOME ERUPTIONS.

Delivered to the Post-graduate Class, Westminster Hospital, May 27th, 1902.

By T. COLCOTT FOX, M.B., F.R.C.P.,

Physician for Diseases of the Skin to the Westminster Hospital.

GENTLEMEN,—I propose to discuss with you today a group of eruptions with which the profession in general is not as yet familiar, and I am not aware that any general account of them has been given in this country, except in a special journal. Nevertheless some of these eruptions are not so very uncommon, and further, a peculiar interest attaches to them because of their supposed connection with tuberculosis. If such a relation be proved these eruptions are often important as indicators of concealed tuberculosis elsewhere.

Allow me to lead up to their consideration by a few remarks on accepted forms of cutaneous tuberculosis. The scientific study of cutaneous tuberculosis, as you know, dates from recent times, and its multiform phases have been reduced to order by modern experimental, histological, and bacteriological study. We occasionally meet with a cutaneous tuberculous ulcer (the so-called *true or miliary tuberculosis*), occurring generally as an auto-inoculation about the mucous orifices secondary to pre-existent visceral tuberculosis, but sometimes as a primary inoculation from virulent bacilli. This ulcer is perfectly analogous to the tuberculous ulcer of the tongue, and displays, firstly, the miliary nodules familiar to us in internal organs whose typical development leads to caseation, breaking down, and formation of ulcers, and secondly, the characteristic bacilli in more or less abundance. *Lupus vulgaris, or nodular or tuberculated lupus*, with its manifold secondary and subsidiary modifications and complications,

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brought about either by differences of site, regional or anatomical, by the varying virulence of the parasite and its mode of implantation, and the reaction of the tissues, is the commonest and must be considered the most characteristic reaction of the skin to the *direct* inoculation of the tubercle bacillus. Lupus is rarely produced in the skin by the medium of the blood-current. The disease not infrequently spreads from the nose, and the skin may be inoculated from deeper-seated disease. When, however, the skin is involved by fistulous tracts leading from tuberculous bones, tendon sheaths, joints, bursæ, lymph-glands, etc., a somewhat different phase is apt to be assumed, often called *Scrofuloderma* (*Tuberculosis colliquativa*, Jadassohn), and characterised by necrosis which is only exceptional in lupus vulgaris. The dermis is said to be, like tendon, an unfavourable soil for tuberculosis, but however that may be, the infective power of lupus vulgaris varies widely in different cases. Lupus may remain localised, or occasionally multiple patches may form by auto-inoculation. Such patches are rarely numerous, and are only symmetrically placed by haphazard, so to speak. Let me ask you to bear specially in mind one evidence of infective power, viz. the evolution around a parent patch, or the reappearance in a scar, of the characteristic little "apple-jelly" satellite nodules, for we must compare these with the eruptions which I shall presently introduce to your notice. Further, let me remind you that, though the histological features are significant, you must not expect to find the classical tuberculous appearances which are displayed in the so-called true tuberculous ulcer. Unna points out that the elementary nodule of lupus consists of a compact mass of cells, known as plasma cells, and these become transformed into a homogenised mosaic of cells, and finally into a group of peculiar giant-cells; but diffuse lupus consists of unaltered plasma-cells without the degenerated elements, and therefore the initial omnipresent and characteristic tissue is the tubercular plasmoma, as he calls it, which does not go on to giant-cell formation, and has no necessary primary connection with a blood-vessel. All combinations of these conditions are found, but remember that you will not always find in diffuse lupus the "areas" or tuberculous "follicles" seen in the true tuberculous ulcer. Koch's bacillus has been found many times, but generally requires laborious search, and so experienced a worker as Darier acknowledges he has not met with it. Analogies may be drawn from tuberculous bones and glands. Lastly, let us remember that for successful inoculation in susceptible animals the lupus tissue has to be inoculated under special conditions as to bulk of material and site of inoculation.

Before leaving this part of my subject, let me draw special attention to two peculiar phases of cutaneous tuberculosis of systemic origin. The first is the so-called *scrofulo-tuberculous gummata* of childhood, the *tuberculomata* of Lannelongue, illustrated by these drawings. They must be distinguished clinically from the gumma-like tuberculous masses in chain formation, which may arise secondary to a tuberculous lesion of the hand, as in these other portraits. Now, although these tuberculomata may occur without evident association with other evidence of tuberculosis, yet concomitant bone and gland lesions are very frequent. They are certainly of systemic origin, and the infective agent is possibly determined to the hypoderm by the nutritive activity of this region in childhood, as at other times to the bones of the hands and fingers, and so on. They are not necessarily symmetrical; they may disappear spontaneously, and they certainly have little infective power. The histological features of tuberculosis are very suggestive, but tubercle bacilli are often difficult to find. My friends, Dr. J. H. M. Macleod and Oliver S. Ormsby, after searching about seventy sections from a case under my care, only found in one section two bacilli. Let us note that the initial changes in this case were in and about the veins.

The second phase is the so-called *exanthematic lupus vulgaris*, or *eruptive disseminated tuberculated lupus*, characterised by the outburst of multiple nodules, clinically like lupus, often following some specific fever in childhood. In the cases occurring after an acute specific fever, such as measles or varicella, the lesions may persist and spread, or disappear spontaneously. Here is a portrait of such a case, which I demonstrated to the Dermatological Society of London. All the elements gradually disappeared except one on a knuckle, which became verrucose. Histologically the structure was of tuberculous type, but bacilli were not found. Apropos of such cases, Darier, who

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has had opportunities of studying several examples, says that though some, proved by inoculation, are certainly tuberculous, others may fairly be included in the category of "tuberculides" about to be considered. Otto Leichtenstern also recorded a remarkable case of *acute miliary tuberculosis of the skin in a case of acute visceral tuberculosis* in a child. The majority of the papules, which were in size from a poppy seed to a hemp seed, firm to the touch and acuminate in shape, underwent complete involution with slight desquamation. Some vesiculated, others became capped with pus. Numerous tubercle bacilli were found in the eruptive elements. I have seen a somewhat similar case, though I was unable to make a microscopical examination.

Now, gentlemen, in recent years a further group of eruptions have been attentively studied, and appear to have a common parentage, and form a natural series. It is thought that these eruptions also have a close relation to tuberculosis. They have been described under a host of names, such as *Lichen scrofulosorum, Folliculites disséminées symétriques des parties glabres à tendance cicatricielle* (Brocq), *Folliculitis exulcerans* (Lukasiewicz), *Folliclis* (Barthélemy), a term designed to recall the follicular origin, whilst distinguishing the eruption from the vast group of folliculites; *Acne cachecticorum* (Hebra-Kaposi), *Acne scrofulosorum* (Radcliffe-Crocker, Colcott Fox), *Acne varioliformis* (Bronson, Grunewald); *Acné miliare scrofuleuse générale* (Bazin), *Acné indurée généralisée* (Aubert), *Acne telangiectodes* (Kaposi), *Acnitis* (Barthélemy); *Hydradenitis suppurativa* (Pollitzer, Dubreuilh); *Lupus erythematosus disseminatus* (Boeck), *Lupus miliaris* (Tilbury Fox), *Lupus follicularis disseminatus* (Finger, Saalfeld); *Érythème induré des scrofuleux* (Bazin), etc. Boeck called them the *exanthems of tuberculosis*, and Darier has suggested as a convenient label, possibly temporary and without prejudging the pathogeny, the term *tuberculides*. This limited use of this term is so far open to criticism that it should be applicable to all cutaneous tuberculosis in an analogous manner to the use of the term *syphilides*. The numerous names given to these eruptions set forth their multiform phases. Each name has been applied to a particular phase, and will not cover all. For example, the eruption is not always developed about a pilo-sebaceous follicle or the sweat glomerulus. Attempts have been made to reduce these tuberculides to some order and classify them, but so far with only partial success.

As I am only able to show you a few patients and not a representative series of these eruptions, I have arranged here my collection of portraits, which I think will give you a fair idea of the character and range of these disorders. I am not going to surfeit you with a description of all the phases described, but let me briefly point out some of the types.

The first type, which is well marked, is *Lichen scrofulosorum*. As you see by these four portraits, the eruption is characterised by the symmetrical evolution of numerous indolent micro-papules, due to a perifolliculitis of little intensity. These papules have a characteristic tendency to group in clusters, and to be distributed over the trunk and limbs, with a special predilection for the lower part of the trunk. The eruption appears suddenly, may last an indefinite time, disappear spontaneously, and reappear. It is an eruption almost special to childhood and adolescence. Recently Jadassohn and Rona have described abortive and evanescent forms of this eruption which are said to occur at all ages. The association with tuberculosis is frequently striking, so that we must agree with Jadassohn that lichen scrofulosorum is a disease of the tuberculous, and not of cachectics generally. Hebra rarely saw it in association with phthisis, but found about 90 per cent. of the patients with enlarged lymphatic glands or periostitis, caries or necrosis, or scrofulous sores, or with enlarged abdomen, presumably due to mesenteric glandular disease. Jadassohn, in nineteen typical cases, found fourteen tuberculous and only one certainly free; and Lukasiewicz observed tuberculous complications in about one half of his forty-three cases. I need not elaborate this evidence.

As to the influence of tuberculine injections, Neisser twice saw typical local reactions, and Jadassohn fourteen times out of sixteen. Hallopeau lays great stress upon his observation of the appearance of satellite lichen scrofulosorum papules round a lupus centre, as we see sometimes miliary papules crowd round a central larger papular syphilide.

The histological picture, on the whole, raises the strongest presumption in favour of the tuber-

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culous nature of the lesions, but let us bear in mind that a somewhat similar picture with giant-cells can be presented by the corresponding micro-papular syphilide.

The search for Koch's bacilli has been unsuccessful, with the exception of a single one found by Jacobi and those recognised by Wolff and Pellizari.

As for inoculation, Jacobi succeeded in producing a local mesenteric glandular tuberculosis in one case, Pellizari was also successful in a special late phase of the eruption described by him, and Wolff got positive results. For the most part inoculations fail, as in Jadassohn's nine cases.

As a connecting link, lichen scrofulosorum has been noted to co-exist with hemp-seed-sized lupus-like nodules, and with so-called acne cachecticorum.

We next come to a crowd of eruptions which it is somewhat difficult to arrange in order. The different phases depend on the bulk of the infiltration (millet seed, lentil, hemp seed, split pea, nut); its origin in the deeper or more superficial layers of the dermis; its situation around the pilo-sebaceous follicle or sweat glomerulus, or elsewhere; the shape and colour of the lesions; the number of elements, and their distribution; the presence of pustulation, necrosis, or ulceration. The difficulty of classification is increased by the fact that the shades of difference between the various phases are often very delicate, and that a given eruption is often made up of several phases.

I think the most convenient course is to regard the prevailing bulk of the eruption, and divide them into papules and nodules, and thus speak of a papular and nodular tuberculide as we do with syphilides. Further, we can signify whether the eruption is scaly, or pustular, or necrotic, or ulcerative. Thus we can speak of small or large, *papulo-squamous*, *papulo-pustular*, *papulo-necrotic*, *nodulo-pustular*, *nodulo-necrotic*, and *nodulo-ulcerative eruptions*.

First, then, let me show you three portraits of cases under the care of Haushalter. Boeck characterises these eruptions as papulo-squamous. This case of Norman Walker's seems to be of similar character. Contrast with these two photographs of the so-called *folliclis* showing necrosis of the lesions and scarring. The eruption is frequently papulo-pustular, rarely approaching the impetiginous type, and generally of an indolent acneiform type. This drawing from one of my cases of so-called acne scrofulosorum illustrates a phase sometimes seen in infants. To contrast with it let me show you this remarkable papulo-pustular eruption on a woman's arm. The pustules have dried up into thick crusts, and the eruption tends to form groups. It is an old drawing, and is labelled *acne cachecticorum complicating lichen scrofulosorum*. A less severe eruption is this grouped, small-sized, papulo-pustular eruption on the back of a delicate girl. I wish, also, to draw your attention especially to this case of widely disseminated papular and nodular eruption in a man with tuberculous disease of the neck glands. Some lesions are slightly pustulating, some necrosing. Another portrait is a *fac simile* of the last. The young man had been operated on several times for old-standing tuberculous glands in the neck. I was greatly puzzled to make a diagnosis of his generalised eruption, and made a wrong one at first, viz. syphilis. Notice the large split-pea- and hazel-nut-sized necrosing nodules on the hips. Sometimes the crowding of elements results in large infiltrated plaques, as shown in these portraits, especially about the legs, where also nodules of greater volume are more frequent. Of much interest are these three illustrations of a peculiar phase found disseminated and localised on the face. Many cases are on record under such different names as *acne-lupus*, *acne telangiectodes*, *miliary lupus*, *disseminated miliary follicular lupus*, and so on. The papulo-nodules are of a somewhat acneiform type with but little tendency to pustulate, and they tend to leave varioliform scars. A striking feature is the "apple-jelly" aspect of the infiltration, like that seen in small lupus nodules, and this appearance has led to confusion with a rare eruption known as *colloid milium*. Let me draw your attention to the fact that two of these portraits show uncoloured shotty nodules on the backs of the hands. (Two living patients with this phase were demonstrated, also a girl with tuberculous glands in the neck and papules on the backs of the hands.)

I have mentioned the large volume of some of the nodules in certain cases. Here is a picture of a disseminated eruption in which all the nodules are of considerable size and project boldly from the skin. Occasionally, however, the nodules are

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deep-seated, and only gradually reach and project the surface. Such is the case in the portrait of the front of the legs of a woman. Still larger eruptions of the same kind are here portrayed on a girl's buttocks and back. The thick crusts covering the infiltrations gave a certain superficial resemblance to ecthyma, but beneath the crusts were punched-out ulcers.

Finally, I come to the eruption described by Bazin as *erythema induratum* of the scrofulous. This eruption has its site of predilection about the calves, especially just below the bellies of the gastrocnemii, but it may be more extensive, though with one exception rarely beyond the legs. That exception is the occurrence of small nodules on the backs of the hands just like those to which I have already called your attention. Females are almost exclusively affected, and perhaps young girls especially. The subjects not infrequently have glandular mischief in the neck, and they have the poor circulation so commonly associated with these tuberculides. The nodules begin in the hypoderm, where they are often better felt in the doughy swollen legs than seen. They behave exactly as syphilitic gummata, and gradually discolour and involve the more superficial skin. They may undergo resolution, or discharge by a small opening, or break down into a punched-out ulcer. Evolving from time to time, or apparently stimulated into excitement at intervals, this eruption may last many years. (A case was demonstrated.)

Histologically we get the same presumptive evidence of tuberculosis, and two successful inoculations have been carried out in guinea-pigs, by Thibierge and Ravaut and in a case of my own. Jadassohn in two cases also found the pus from the fistulæ rendered guinea-pigs tuberculous. In two typical cases also he obtained a local reaction with tuberculine.

To complete the subject I should mention that distinguished authorities regard *lupus erythematosus* as belonging to the same category, but there are strong arguments against this view, and I will not enter into a discussion now which has already been fully debated.

When we consider these eruptions as a whole we find they have certain clinical characters in common. The elements are variable in number, generally numerous, sometimes copious. They are often isolated, sometimes grouped in figured patterns, or crowded into patches. Experience teaches us to appreciate certain peculiarities of aspect. In size and location in the skin the elements may vary from micro-papules to voluminous nodules. Whether they pustulate, or necrose, or ulcerate, they always display the initial cellular infiltration as in syphilides. They usually appear brusquely without fever; in crops sometimes subintrant and continuous, sometimes intermittent, and the eruption may be comparatively temporary (weeks or months) or last on and off for years. Individually the elements are indolent without much disorder of sensation. They tend to disappear with or without consecutive atrophy, suppuration, necrosis, or ulceration, and a cicatrix is frequently left. Different forms have a marked predilection for special regions, and can be localised there. Any part of the surface may, however, be involved, and the eruptions may be generalised. They are bilateral, with a notable tendency to symmetry.

Now the strong disposition to attach these eruptions to tuberculosis is influenced by clinical, histological, and experimental evidence, which is stronger for some phases than others. In a considerable proportion of cases the concurrence of tuberculous lesions of other parts is manifest, though common phthisis is not frequent. In quite a number of patients the family history points to susceptibility to contract tuberculosis, and the patients themselves are of a type which we recognise as prone to the disease. In some way we may even strongly suspect a latent or attenuated tuberculosis. It must be confessed, however, that occasionally the subjects are, and have been, apparently in good health. Visceral tuberculosis has declared itself at a late stage of the eruption or subsequent to it, but perhaps it is wise not to press this argument too far.

With regard to the histological evidence Darier, who speaks with authority, says that one can class the lesions in a continuous series, which unite by insensible degrees two types in appearance opposed. In type A there is a new formation in the dermis of a characteristic tuberculous tissue with numerous giant, epithelioid, and lymphoid or plasmatic cells, these elements affecting a more or less nodular or "follicular" grouping, and having a tendency to undergo caseous degeneration. In

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type B the lesions consist essentially of round or plasmatic cells ensheathing the vessels of the dermis; in vascular alterations with dilatation, or, on the contrary, constriction and even obliteration of their calibre; and in the production of more or less extended necrotic areas. The justification for uniting these two types seems to lie in the delicate transitions, and in the concurrence of the two in the same case. The prominence of vascular lesions I have already referred to when speaking of scrofulo-tuberculous gummata.

Nevertheless, in the absence of Koch's bacilli the histological evidence can only be presumptive, however strong the presumption.

Now, observers have nearly always failed to find the bacilli, but I have already insisted on the perseverance necessary to detect them in many cases of lupus vulgaris and the scrofulo-tuberculous gummata of childhood. What significance is to be attached to the find by Jacobi, Pellizari, and Wolff in lichen scrofulosorum remains to be proved by further research.

Inoculations in guinea-pigs have almost always failed, but here again we must remember that the bacilli may have disappeared in old lesions, that the lesions may be caused by dead bacilli, and that for successful inoculation, even in lupus vulgaris, according to Leloir, a quantity of tissue must be introduced which is relatively considerable. However, let us note that Jacobi, Pellizari, and Wolff have been successful with lichen scrofulosorum; Haushalter and Lefebvre in their papulo-squamous tuberculide; and Thibierge and Ravaut, myself, and Jadassohn in erythema induratum. Injections of tuberculine may not, perhaps, be regarded as a test of high value, the effects are apt to be irregular and inconstant in tuberculosis, and other lesions react. However, Jadassohn was successful in fourteen out of sixteen cases of lichen scrofulosorum, and in two cases of erythema induratum.

Now we must admit, I think, that some systemic exciting agent is necessary to explain the dissemination and symmetry of the eruption, though those observers who regard psoriasis as a local parasitic disease might object to this view. We cannot at the present date fall back on the old conception of a scrofulous diathesis as a sufficient explanation. Various hypotheses have been put forward. It has been suggested that some unknown microbe may be operative, which demands a soil prepared by Koch's bacillus. Hallopeau and Boeck favour the view that the eruption is due to the toxines disseminated by Koch's bacillus rather than to the microbe itself, and these would act sometimes on certain vaso-motor centres, or directly on the skin itself. Hallopeau thinks the rare finding of a bacillus not incompatible with this theory. In this connection Boeck lays stress on the primordial vascular and perivascular alterations which, he thinks, distinguish certain tuberculides from the common forms of cutaneous tuberculosis. The evolution of lichen scrofulosorum under the influence of an injection of tuberculine in one case is cited in evidence, to which the objection is raised that this injection may have simply excited a latent eruption into evidence, as I have seen it do in nerve leprosy. *A priori* we should incline to the view that the toxines would excite the evolution of erythemata as the diptheria antitoxin and tuberculine injections do.

A third hypothesis which, perhaps, finds most favour supposes that the eruption is due to bacillary emboli of varying virulence conveyed by the blood-current. If so, the bacilli are probably scanty as a rule, of little virulence, and rapidly succumb, or are already dead ones. Darier considers this hypothesis adequate to explain all the facts, histological and of other kinds, known to us. If confirmed by subsequent observations these tuberculides will be absorbed into the common group of cutaneous tuberculoses.

In conclusion, allow me to call your attention to the analogy which exists between these tuberculides and syphilides. I will not strain the point too far, but I think you will agree with me when you look at these portraits of syphilides which I have arranged for comparison side by side with the tuberculides.

Prolapse of the Urethra.—A case of prolapse of the urethra in a healthy and well-developed girl of five and a half years is reported by Bente ('Münch. med. Woch.,' No. 53, 1901). The common predisposing factor—general debility and relaxation—certainly could not be evoked to account for the causation of the trouble. It seemed dependent upon constipation causing straining at stool, and upon coughing. Medical treatment was unsuccessful, and operation was required, and gave good results.—*Amer. Journ. of Obstetrics*, July, 1902.

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TWO CLINICAL LECTURES
ON
"UTERINE DISPLACEMENTS."

Delivered at the Medical Graduates' College and Polyclinic.

By W. J. GOW, M.D.,
Physician-Accoucheur to Out-patients, St. Mary's Hospital, and Physician to Queen Charlotte's Lying-in Hospital.

LECTURE II.

GENTLEMEN,—I want to speak to-day of those forms of displacement which are rather alterations in attitude than alterations in position, that is to say, those displacements which are generally spoken of as anteversion or retroversion or retroflexion. The improvements in the methods of pelvic examination, especially bimanual examination—which is of comparatively recent introduction—has led many observers to attribute the symptoms complained of by women to anatomical, rather than to physiological causes. Changes in the position or attitude of the uterus are common enough, and easily made out on physical examination, and offer an apparently ready means of explaining the aches and pains of which so many women complain. An ill-nourished nervous system, or ill-nourished muscles and ligaments, account for many a backache. The temptation to assume that an altered position of the womb, which may sometimes be found to exist in such cases, is the cause of all the symptoms is no doubt great, but none the less it must be resisted, because often the assumption is quite wrong. Now, the womb maintains its position in the pelvis by virtue of several things; for example, the utero-sacral ligaments, the vesico-uterine ligaments, the broad ligaments, and the round ligaments, all play a part in maintaining it in the ordinary position. And there is another factor, undoubtedly, which plays an important part, and that is the natural tonicity of the uterine muscle. The uterus stands up, it is not held up, and it stands up in a particular attitude, that is to say, looking forwards; and this attitude is as much due to its muscular tone as to anything else. Of course the tonicity of the vagina plays an important part in holding it up from below, but we must not forget that the uterus stands up also. If that is so, it is obvious that the attitude of the uterus will depend very largely upon the condition of the muscular tone; and I think one of the first things that should be recognised is that, even in health, the uterus does not always occupy the same position. I am not speaking of the constant alterations of position that it undergoes as a result of the condition of the bladder. The uterus is pushed on one side by every organ; upwards and backwards by the bladder as the bladder fills with urine, and from one side to the other by the rectum. But whether it stands firm and straight, or flaps backwards, depends to a great extent on the muscular tone of the uterus at the particular time. There is no doubt that in certain people the uterus is not always in the same position. A uterus which on the first examination may be found to be directed backwards, may, a week later, or possibly even a day later, be found in the more ordinary attitude of anteversion and flexion, and this without any form of treatment whatsoever. It is therefore important to realise that a woman's uterus is not always standing up firm and stiff, but that sometimes it has its moments of relaxation, at which times it often falls backwards. And thus you can easily see how you might misjudge a fellow practitioner who has stated that a certain patient's womb looks backwards, whereas you yourself find that it looks forwards. Of course it is possible that one or other observer may be in error, but, on the other hand, it is quite possible for a uterus which is lying back in the morning to be straight up in the afternoon; that point I am quite certain of. There are certain analogies between the bladder and the uterus, and if you look inside the abdomen of a woman in the post-mortem room you may find that the bladder, if empty, will be in one of two conditions: it may be perfectly flaccid, or, on the other hand, it may stand up firm and stiff like a uterus. In one condition it is said to be in a state of diastole, and in the other in a state of systole. It is not unreasonable to imagine that the uterus in like manner may be sometimes in a state of systole, and at other times in a state of diastole. Besides, on looking inside the abdomen after death one finds the uterus in one of these two conditions: either standing up stiff and firm, if the patient happened to die when the part was in a state of systole; or, on the other hand, it may be found flaccid and limp, with the fundus directed towards the sacrum. In such a case the uterus may be said to be in a

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state of diastole with the fundus looking backwards. I think that is true, that during life the uterus is not always in a state of systole. In a normal state, where there is tonic contraction and where the muscular tone is good, the uterus will stand up. At other periods of time it may be in a state of diastole, and then it often lies directed somewhat backwards. This illustrates the importance of muscular tone as a means of keeping the womb in position, and that the uterus stands up and is not held up.

Of anterior displacements of the uterus I shall not say much. The term displacement itself is an ugly one; it suggests to the patient some terrible condition. We know that it is usual for the uterus to look forwards, and for a certain degree of bending to exist at the junction of the body with the neck of the uterus, or thereabouts. That condition, no doubt, in many cases may be somewhat increased; in other words, the degree of it may be greater than usual. And the flexion, at any rate, is likely to be greater than usual in those cases—which are not uncommon—where the uterus has undergone imperfect development, a condition which may be termed, if you like, infantile uterus, a condition of affairs which I think is very much more common than is often thought. Many of these cases, which are dubbed cases of anteflexion accompanied by sterility, are cases of an immature womb which has assumed that attitude. It is a very common thing, as a result of congenital failure of development, for this flexion to be exaggerated. In other cases, as we shall see later on, the ill-developed uterus possesses a curve in the opposite direction, that is to say, the angle of flexion lies on the opposite side, and the uterus is thus retroflexed instead of anteflexed. A sharp degree of anteflexion or retroflexion in a sterile woman is often highly suggestive of incomplete development of the part.

Of anteversion, pure and simple, I think nothing need be said. I have never been able to discover that definite symptoms can be attributed to it, and I think we may safely say there is no pessary in existence which diminishes the amount of bending forward of the uterus.

As to backward displacements, these are, I think of more common interest; but you must remember that there are several varieties of this condition, and two in particular which I think should be distinguished from each other, but which I think often are not so distinguished. The least common is the one in which the uterus looks backwards and is in a state of more or less complete flaccidity; that is to say, with the fingers or with the sound the uterus can be raised up and made to look forwards, and not only raised up and made to look forwards, but made to stop in that position. In articles on this subject in the text-books you will see various pictures in which the sound is shown introduced into the uterus, and the uterus is thereby raised up and replaced, the authors forgetting that in the common form of retroversion or retroflexion directly the sound is taken out the uterus dives back into the original position it occupied, or at least approximately so. On the other hand, in the atonic form, if you pass a sound you can raise the uterus up, and can move it in any direction you choose, or you can catch it between your two hands, and antevert it or retrovert it at pleasure. The uterus is in a state of diastole. This form of retroversion is not common, or at any rate not nearly as common as the other; it is not the form which usually follows childbirth or miscarriage. In the common form the uterus appears to have adopted the backward position in the early days of the puerperium, and seems to have "set" in this position of retroversion or retroflexion whilst involution was in progress. The result is that all the other structures in the neighbourhood of the uterus adapt themselves to this new position; and in addition the natural stiffness of the uterine tissue prevents the sudden conversion of a uterus from the attitude of retroflexion into the attitude of anteflexion. It is just as possible to convert such a uterus as that suddenly, by means of the sound or by the hands, into an anteverted and anteflexed position as it would be to convert the normally placed uterus into a retroverted and retroflexed one. By means of a sound, a normally situated uterus can be tipped backwards, but it will resume its normal position directly after the withdrawal of the sound. It is equally impossible to alter the position of the uterus suddenly in this form of retroversion by means of manipulation with the fingers or by the introduction of a sound. The uterus may be raised up with the sound, or it may be made to look forwards, but directly the sound is removed it returns to its former position. This is the commonest form of backward displace-

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ment that is met with, though the flabby form is met with from time to time.

I have spoken so far of backward displacements in rather a vague way as implying a retroversion; but when the uterus looks backwards, very often the angle between the body and the neck is concave behind instead of being concave in front, that is to say, the uterus is bent backwards at that point instead of being bent forward, and thus the uterus is often retroflexed as well as retroverted. A great deal of importance has been attributed to the retroflexion. It is difficult to see why, because we know that normally the neck and the body of the uterus do form an angle, and at first sight it is difficult to see why, if that angle looks backwards, there should be any particular difference to the patient compared with when it looks forwards. It is not as if the neck and the body of the uterus ought to be perfectly straight; they ought not to be, and generally they are not. At any rate, this condition of retroflexion mostly accompanies retroversion. It does not occur in so marked degree when the uterus is flabby and lax, as in the first class of cases I have described, at any rate the angle formed is not so acute. In the class of case which results from childbirth, very often the angle is formed behind instead of in front. On the other hand, as I have said, retroflexion of the uterus is not uncommon as a congenital condition, and is often associated with imperfect development.

Next, I would insist that retroflexion is a condition which is perfectly unalterable by any pessary introduced into the vagina, and those who believe in the treatment of retroflexion will admit the same thing. The only form of pessary which can possibly overcome this condition is the intra-uterine stem pessary, and though sometimes such a pessary may be used without calamity it is a source of great anxiety as long as it is retained. I shall speak of this further in discussing the treatment of this condition. Remember that as regards the causation of these conditions there are certain congenital forms, and there are also certain acquired forms. Remember, further, that in the congenital cases the pelvic organs often exhibit signs of imperfect development. Under such circumstances it will often happen that in the earlier period of puberty there is not, or may not be, much discomfort complained of. But a uterus which is ill developed is one which generally fulfils imperfectly the menstrual function, and not only fulfils it imperfectly, but with much pain and discomfort to the individual. Such cases come under one's notice because of dysmenorrhœa or scanty menstruation. If the pain is of extreme intensity and is unrelieved by medicine, you make an examination and you find the uterus retroflexed. You will bear in mind what I have said, and you will realise that that is probably simply an indication of an imperfect development, and that she is suffering in this way as a result of an imperfect development, and not because the uterus is inclined at this particular angle. On the other hand, you may find it sharply anteflexed, and you might be inclined to explain the symptoms on the ground that the uterus is bent in this manner. Again, you may remember that that condition is often assumed by imperfectly developed uteri, and this leads to the belief that the dysmenorrhœa is rather of developmental origin than that it is due to the flexions or versions. Now, if such a patient marries, what will be the probable history? In the first instance, she will be sterile. This is not due to the fact that the uterus is retroflexed, but to the fact that it is ill developed. And sterility brings in its train, as probably all of you know, a large number of neurotic symptoms. The fact of not becoming pregnant preys on the patient's mind, and it is regarded by her as a sign of some disease. The result of that state of mind is that she develops a more severe form of dysmenorrhœa than she had before, and dyspareunia, if not present from the first, often develops; and, thirdly, she complains of pelvic pains and backaches and other symptoms, all of which she attributes to the condition of the uterus. She also usually exhibits marked depression of spirits, imperfect digestion, imperfect nutrition and assimilation, and, as a consequence, very often wastes. If care be not taken you may, as a result of examination, say here is retroflexion, and this is the cause of the troubles. In reality it is nothing of the kind. The bulk of the symptoms are undoubtedly neurotic, and they have followed upon the absence of conception. It is difficult to realise to the full extent the troubles which absence of conception gives rise to in married life. But sometimes in patients with imperfectly developed uteri you will find, even though they may have had no dysmenorrhœa

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before, they develop it after marriage. That these symptoms persist in spite of various operative measures that I shall speak of, I know to be a fact. I have seen patients in whom, owing to the fact of their having various abnormalities, I have set to work to remedy them, and I have remedied them all, but the symptoms have persisted.

The acquired cases, as I have already said, generally follow childbirth or miscarriage. It has been often supposed that falls, blows, and injuries of various kinds lead to uterine displacements. I think I may fairly say, as I have been on the lookout for such cases, that I have never met with any patient who displaced her womb by any injury, and I think I may further say that a woman with a normally placed womb is entirely unable to displace it by any known form of accident. Remember this is very important, for the reason that patients are undoubtedly impressed with the view that any strain or overreaching, or any carrying of heavy weights, which acts are often attended with sudden muscular pain, may cause displacement of the womb, and that the pain which may persist after these acts is due to this cause. These patients come fully prepared to be told that the symptoms which they complain of are the result of some displacement of the womb. As a matter of fact, every one sees many cases of this kind, and in the great majority of such cases the uterus will be found occupying the usual position. I believe that the only circumstance in which a womb could be altered much in position by a great strain or fall would be where the womb was already in a position of retroversion or retroflexion. If the fundus lay at the entrance of the Douglas's pouch, close to the utero-sacral ligaments, the sudden strain or fall might drive it down more deeply, and might thus give colour to the view that the fall or strain had displaced the organ. But I am sure that a uterus normally situated, standing up and looking forwards, cannot be converted into a uterus looking backwards by any form of injury. It is often a matter of the greatest possible difficulty to persuade such patients that they have not displaced their womb in this way. You know that men as well as women, after injury to the spine, or after a wrick or a sprain, are sometimes convinced that some serious disease has set in, and thus their pain persists. A pain which was ligamentous and muscular in the first instance, and which would have disappeared in the ordinary course in a few days, may persist for weeks or months, and is due to neurosis. I think, therefore, we may eliminate from the causes of backward displacement any injury or sprain, because unless the uterus was originally looking backward, no amount of strain or injury will make it do so.

The symptoms which are generally ascribed to this condition are well known. They are pain in the back, pain in the iliac fossa, pain often described as of a bearing-down character. When you come to think of it, there are very few classes of cases which come into any gynæcological department where the women do not complain of three things: pain, generally in the left iliac fossa and sacral region, discharge, and bleeding. Those are the symptoms which occur in nearly every woman. Pain is an extremely common complaint of women, and the pain is oftener referred to the left side than to the right. The bearing-down sensation is not necessarily associated with descent, and though every woman who has bearing-down pains thinks such symptoms must be dependent upon the condition of her womb, it by no means always is so. In addition to that, although they do not complain of it, if you ask you will find that they are generally constipated, and they are frequently ill nourished. The diet of women, especially of the poorer classes, is bad, and the digestive powers are feeble; moreover many such women lead a life which is productive of or conducive to ill-health, compared with the male sex. These women get pain in the left iliac fossa in the back. That is to say, these patients get pains in the positions where there is a good deal of constant strain. After all, the weight of the body is supported by ligaments situated in the sacral region, and, no doubt as a result of bad nutrition, they suffer from the strain to which they are exposed. This aching of ligamentous and muscular structures is interpreted by the woman to mean that there is pressure upon the womb, or that it is in an abnormal condition. No doubt the muscular structures of the abdominal wall are exposed to a good deal of pressure from the intestines, which are driven down to that part of the abdomen owing to the constriction of the upper part. The muscular walls are often overstretched as a result of pregnancy, and, owing to lack of tone, imperfectly support the abdominal contents.

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This is a source of pain or discomfort, and also of flatulence. Even if in such cases the uterus does look backwards, this fact is by no means a complete and adequate explanation of the symptoms. You will find that in many such cases, if no treatment is directed to the womb, and if the general nutrition of the patient be improved, and her bowels are regulated, and she is, in addition, put on iron, which comes amiss to very few women, and if, in addition, she is dieted on more rational principles than she has been used to the pains will lessen or disappear. Moreover, you will often find women who are in good general health with the uterus looking backwards, and yet none of the above-mentioned symptoms are present. Of course, a uterus that is looking backwards may be a healthy uterus, or, on the other hand, it may be an unhealthy one. Inasmuch as this alteration of position often directly follows childbirth, it not uncommonly follows that a patient with a retroverted uterus may complain of menorrhagia or metrorrhagia, but this is not because of the retroversion, but because of subinvolution. The term subinvolution is a convenient word which covers different classes of cases. I take it that the word subinvolution means that the uterus is larger than natural, either—and this, I think, must be rare—because the process of involution has not been completed, or because a certain amount of chronic inflammatory changes in the connective tissue of the uterus followed the childbirth. Of these two conditions, the latter is often called chronic metritis or chronic uterine hyperplasia, and the other true subinvolution. Probably in most cases in which the uterus remains bulky with thickened walls after childbirth or miscarriage, the condition is one of chronic interstitial hyperplasia; infective changes have occurred in the mucosa, and have spread thence to the uterine parenchyma, and this has led to an increase of connective tissue and to general thickening, a thickening which may remain permanent. This condition often leads to a greater loss at the menstrual period. If the uterus has involuted properly, and no interstitial changes have occurred, the uterus may be found retroverted and retroflexed, and yet there are no symptoms at all. The important question is whether the retrodisplaced uterus is or is not prolapsed. In many cases the uterus looks backward, and no marked descent may occur. If the uterus is healthy and properly involuted, I am sure no definite symptoms dependent on this condition exist. If, as is sometimes the case, the utero-sacral ligaments are relaxed and the uterus plunges through them and descends deeply into Douglas's pouch, a certain number of women experience discomfort, and complain of a bearing-down sensation, which is apt to be attributed to the retroflexed position of the uterus. It is really due to the accompanying descent or prolapse. This condition of affairs is, in the vast majority of cases, treated by the use of a vaginal pessary; but all that a vaginal pessary will do is to raise the level of the uterus, a fact which you can make out for yourselves with the greatest possible ease in any case. A uterus which is retroverted and retroflexed can be pushed up, provided there are no adhesions, and they are very uncommon, above the level of the utero-sacral ligaments. But it cannot, unless it is quite flabby, be made to look directly forwards. It may be made to occupy a higher level, and can be kept at that higher level. But you find that, although the retroflexion or retroversion persist, the patient now feels more comfortable; she does not feel the bearing-down sensation as she did before. By the use of vaginal pessaries you can *lessen* the degree of retroversion, and you can *do away with* the descent, but you cannot alter the retroflexion. Remember that in a weakly woman who has recently been delivered of a child, or been the subject of a miscarriage, the symptoms of pain and discomfort of which she complains are often the result more of the condition of her general health than of any abnormal position of the uterus, and that good food and tonics do more for her than pessaries.

There are numerous points which one might discuss. But this much deserves to be said, namely, that in women, say neurotic women—and I think you may take it that all women are neurotic, more or less—you can, as an effect of suggestion, produce very often deplorable results. There are a certain number of people walking about with what they are pleased to call a twisted womb. They do not find that out of their own initiative; they have been told that by a medical man, and that that is the cause of their trouble. Sometimes they have no symptoms at all, and in other cases they are merely suffering from chronic dyspepsia and constipation. I have known patients who

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have worn pessaries for years, but all the time the uterus has remained retro-displaced. This goes to prove that the treatment adopted was either highly unsatisfactory or else altogether unnecessary. Patients who have once become firmly impressed with the idea that their womb is twisted are very difficult to cure, I think there is no question whatever that it is therefore a matter of very great importance to avoid, as far as possible, speaking about alterations in position as if they were of very great import. I think if one finds a uterus in a position of retroversion and retroflexion, one has to tell the patient or her friends, more or less in self-defence, that her womb is not quite in the usual attitude, but that it is not in a position which will be injurious, and that it is not one which will give rise to any particular disturbance. Then, if such patients go to somebody else, and he tells them they have a twisted womb, they cannot come back to you and say you had not discovered it; it is well to tell them that it is not a matter of very great importance, and that, as long as there is no descent, it is not one which requires any local form of treatment. It is obvious that congenital retroflexions or anteflexions which occur in virgins, associated with an ill-developed womb, could not be more improperly treated than by means of pessaries.

You may say, in view of what I have said, that the question of operative treatment can hardly arise. But it does arise. I have pointed out that in those case of retroversion or retroflexion with descent following childbirth or miscarriage, relief can be achieved by a pessary, and although all kinds of pessaries have been used, there is no doubt that the ordinary rubber watch-spring ring in the majority of the cases fulfils the purpose better than any other form. But in certain patients the vagina is not suitable for pessaries, and although the symptoms may be relieved, the patient suffers subsequently from irritation produced by the pessary. Again, some patients are very averse to wearing a pessary for any period of time, and in some of these cases it is not necessary, if used at all, to use it for a long time, especially when there is marked descent.

What are the operative procedures? In the first place the fundamental feature of all the operations is that the descent is overcome, the condition which causes the real symptoms, and incidentally the uterus is placed in a position of anteversion instead of retroversion. There are some cases in which the risk of the operation is quite out of proportion to the benefit likely to be produced. There is, for example, the operation of ventral fixation, or fixing the uterus, which is retroverted and retroflexed and prolapsed, to the anterior abdominal wall. In the first instance the union is a good one, but later on it is apt to become a band-like connection, and the uterus may thus fall again into the pelvis. In some cases the union remains good and further descent is prevented. There is another operation, that which is called vaginal fixation, where the anterior fornix is opened, the anterior vaginal wall incised, also the vesico-uterine pouch, and the uterus drawn out and ultimately fixed to the upper part of the anterior vaginal wall. There is another which is generally known as Alexander's operation, which I think is generally improperly described, that is to say, it is not described in the way in which the author intended it to be. It consists, among other things, of shortening the round ligaments. It is obvious that there is no wide field for any operative treatment of this condition, though there are undoubtedly some cases in which operations do good. When operation is desirable the choice lies between those three I have named.

With regard to the vaginal fixation, there are certain advantages in that operation in suitable cases. By suitable cases I mean those in which the likelihood of recurrent pregnancy is not great. Although it is quite true that in certain cases after this operation has been performed pregnancy has occurred and has been accomplished in the ordinary manner, it has happened that when this operation has been done miscarriages have occurred more frequently than they previously did; and if pregnancy has continued, in rare cases delivery has been attended with considerable difficulty. So where pregnancy is likely to occur and the patients are comparatively young, perhaps vaginal fixation is not the best operation to choose; at any rate, if it is done, great care must be taken to leave the fundus free. Vaginal fixation has the advantage of being comparatively easily performed, and it leaves no scar. It consists of opening the anterior fornix by means of a transverse incision. The loose connective-tissue space between the supra-vaginal cervix and the bladder is opened up with

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the fingers and with the knife, and then the utero-vesical pouch of peritoneum is seen and can be incised without any great difficulty. When that has been done, and a free opening has been made into the peritoneal cavity, the uterus has to be seized as high up as can be reached with the volsella. This can hardly be made to reach beyond the os internum, but when traction is made at this point you can grasp it with another volsella at a higher level, and can, as it were, climb up the uterus, until before very long the fundus of the uterus is drawn completely out through the opening which has been made. This operation affords a very good opportunity for exploration of the tubes and ovaries. In some of these cases the tubes and ovaries are found to be in an unhealthy condition, and there is afforded an opportunity for removing one of them if necessary. When that has been done the sutures are passed through the uterine body; and it is important to remember that these sutures should be only passed just above the level of the os internum, and then the sutures should be passed just through the upper part of the anterior vaginal wall, and the uterus pushed back and the sutures tied, so that the lower part of the anterior uterine wall is held in contact with the edge of the anterior vaginal wall, so that it ultimately forms close adhesions and remains permanently in that position. There is no doubt that operation will result in the uterus being kept permanently directed forwards. The sutures should never be introduced high up in the uterine body, but always just above the level of the os internum, so as to give an opportunity for the rest of the body to develop if pregnancy should occur.

On the question of ventral fixation I shall say but little. An incision large enough to admit two fingers is made in the middle line, and the fundus is brought up to the incision and sutured there. Although in the first instance the uterine fundus is in direct contact with the abdominal wall, it does not usually remain so. Adhesions form, and they become pulled on and stretched out into a longer or shorter band. This operation enables one to explore the condition of the uterine appendages and deal with any abnormal condition which may be found. It is more especially suitable in certain cases of direct or utero-vaginal descent.

The operation of shortening the round ligaments is often wrongly described in the English text-books. One gets from such books an impression that the round ligaments are exposed, and traction is made upon them, and thus the uterus is first of all dragged into position and then kept there. I have no doubt that the originator of the operation did not intend that, because he himself persists that it is absolutely necessary in all these cases first to put the uterus into proper position, and then to retain it by this operation of shortening the round ligaments. That, of course, precludes any risk of undertaking such an operation in a case where the uterus happens to be fixed from adhesions, because then it cannot be first put into position. It may be put into position and partly held there by an ordinary vaginal pessary, but the originator of the operation suggested and carried out the plan of first introducing a stem pessary attached to the Hodge into the vagina. This held the uterus forward. The patient was put to bed with it in that position, and then the round ligaments were dealt with subsequently. That point, I think, is not generally insisted upon in the ordinary text-book descriptions of the operation. But that is what the operation really consists of. It has some advantages, perhaps, and occasionally may be of benefit. But it does not permit of any exploration of the ovaries and tubes. It is an operation of which I have a very limited personal experience, and therefore I shall not discuss it further.

Scarlatinal Nephritis.—Baginsky ('Jahrbuch für Kinderheilkunde,' vol. liv, No. 5) observed eighty-eight cases of scarlatinal nephritis in his hospital during the last five years. The earliest day of the scarlatina on which the nephritis developed was the sixth, and the latest was the thirtieth. The majority of the cases began with fever, and those with marked fever proved to be especially dangerous. The quantity of urine was increased at first in some cases. In eighteen children a chronic albuminuria remained, and in five of these unmistakable signs of chronic nephritis developed. Cases treated in the hospital from the onset ran a decidedly more hopeful course than those admitted late. The cause of this is the treatment, consisting of four weeks' rest in bed and two weeks' absolute milk diet. Venesection is recommended for uræmia, and tannin for long-standing hæmaturia.—*Amer. Journ. of Obstetrics*, July, 1902.

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TUMOURS OF THE KIDNEY.*

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LECTURE I.

THE pathology of tumours of the kidney is still to a great extent an enigma. Until recent years but scant attention was given to any serious study of the origin, character, and relationships of renal growths. The uncertainty of the past and doubt of the present are only too evident in much of our current nomenclature. Modern research has, however, thrown some new light on the histogenesis of these neoplasms, and has gone far to indicate means for a more scientific grouping.

In these lectures I shall endeavour to place before you the more important conclusions arrived at, and to indicate lines along which further advance may be made.

Unfortunately most of the old records concerning renal tumours are comparatively useless for scientific purposes from the lack of thorough pathological examination. It is only by a complete investigation of every case that sufficient reliable data can be accumulated to allow of a classification which may indicate not only the morbid characters, but also their respective clinical features.

It is most desirable that we should be able to ascertain with some near approach to accuracy during life the exact nature of the growth, and be in a position to form an opinion regarding the advantages of removal as a means of retaining life or retarding death.

So long as we remain in ignorance of the precise nature of renal growths and the causal agents instrumental in their development, classification is necessarily imperfect, and any grouping adopted must be recognised as merely tentative. A perfect histogenetic arrangement is at present impossible, for such would necessitate a complete acquaintance with the life history of each form of growth. It is desirable, also, that our pathological grouping should be in accord with clinical facts and of practical service to the practitioner. At all events it is needful to avoid hampering progress by the continuation of a loose, uncertain, or misleading terminology. We should aim to secure a grouping which shall indicate: (1) origin; (2) structure; (3) relation to age; and (4) local and general tendencies of the growth.

Special insistance has been made by some on a grouping into those of congenital origin and post-congenital origin. Then, of course, we have the useful clinico-pathological division into benign and malignant.

At the present time our chief guides must be histological and clinical. Over-eager and hurried attempts to secure a mere classification are likely to hinder advance rather than help it. We must be content for a while to collect, investigate, and correlate. But for purposes of convenience I venture to place the following table before you as indicating to some extent the more important groups of tumour formation met with in connection with the kidney and its immediate neighbourhood, and which we are about to consider:

1. True renal tumours—
   - A. Originating in the connective tissues:
     - (*a*) Benign . . . Fibroma, etc.
     - (*b*) Malignant . . Sarcoma.
   - B. Originating in the renal epithelium:
     - (*a*) Benign . . . Adenoma.
     - (*b*) Malignant . . Carcinoma.
2. Pelvic tumours, generally a malignant papilloma (epithelioma).
3. Capsular tumours, generally sarcoma.
4. Hilum tumours, generally sarcoma.
5. Tumours arising from adrenal inclusions.
6. True adrenal tumours:
   - (*a*) Benign . . Adenoma.
   - (*b*) Malignant . . Carcinoma.
7. Perirenal tumours:
   - (*a*) Fibro-lipoma.
   - (*b*) Sarcoma.

Before discussing the special characters of the different forms of renal tumour it will be well for us to remember certain general points. Primary forms are met with in both sexes and at all ages. They constitute, judging from my own experience, about 0·5 per cent. of the cases submitted to autopsy in a general hospital, and form from 2 to 3 per cent. of all primary malignant disease as met with in the post-mortem room. Secondary renal

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growths occur, according to the results of my analysis, in about 8 per cent. of cases of carcinoma and in over 14 per cent. of sarcoma. Unfortunately it is almost impossible to come to any reliable estimate as to the relative frequency of the different forms of growth. In young subjects they are generally considered to be of a sarcomatous character, but many present a complex structure which, undoubtedly, in the past has given rise to much confusion in nomenclature, and concerning which even still there is much difference of opinion.

We are almost completely in the dark regarding the causation of tumours of the kidney. Faults in development have long been considered important factors in their formation. Defective segmentation of the protovertebral somites, development of heteroplastic remnants of the Wolffian bodies, and proliferation of adrenal inclusions have been held responsible for the origin of these tumours, especially those which are congenital or arise early in life. There are certainly grounds for believing that embryonic inclusions may remain dormant for many years, and then, under circumstances concerning which we are in almost complete ignorance, awake to active growth. Heredity, as far as can be ascertained, is a factor of no direct importance. Mechanical irritation, as from trauma or the presence of calculi, has been suggested as of some influence, but of this there is no satisfactory proof. There is no evidence to show that inflammatory or other morbid processes exert any direct influence in initiating the development of renal tumours. There is no satisfactory evidence to suggest that these growths arise from parasitic invasion, and the fact that no small number are undoubtedly of congenital origin is strongly opposed to such a view.

As to the agencies by which imperfect differentiation and irregular or excessive growth of or in connection with the renal tissues is brought about, we have no precise knowledge. Renal tumours are met with in both sexes, at all ages, and in every rank of society. They are also known to occur in the black races. Most authors say that these growths arise more frequently in the male than in the female, but my researches would tend to show that, taking cases at all ages, there is practically no difference. Under ten years of age the female is apparently somewhat more liable than the male. The influence of age is most marked. According to my analysis of cases, over 52 per cent. originate below ten years of age, and over 46 per cent. below the age of five. Renal tumours arise during intra-uterine life. They have been met with in the seven months' fœtus. Not infrequently they are observed clinically within the first two years of life. In many cases where the tumour develops during fœtal life or early childhood, both kidneys are involved, and this strongly points to a simultaneous development. The tumours may be of almost any size. The sarcomata often reach immense dimensions. The average weight of twenty-four sarcomata, from patients whose average age was three years, I found to be over 124 ounces. The so-called "cancers" in adults are usually much smaller. In many instances the tumour long remains reniform. The differences in colour depend in great measure on variations in vascularity and the degree of degenerative change. Great differences occur in the consistency of these growths. Generally speaking, the sarcomata and malignant cystomata are soft and vascular, while the adenomata and carcinomata are often firm and sometimes dense.

Renal tumours often present secondary changes. Degeneration processes commonly occur. Occasionally slight calcareous infiltration is found. Hæmorrhage, especially in the soft vascular forms of sarcoma is not infrequent. Local necrosis may be met with.

There is much difference in the rate of development. Usually the more embryonic they are the more rapid the progress. In children the rate of increase is often particularly rapid. A carcinoma usually progresses more slowly than a sarcoma, and some forms of malignant adenoma would appear to exist for a considerable time.

In considering the different forms of renal growth it is convenient to retain the customary division into: (*a*) tumours of a connective-tissue type; (*b*) tumours of an epithelial type; but it is very necessary to insist that such conventional grouping must not be allowed in any way to fetter us in our consideration of the true nature and actual relationships of these growths. It is certain that complex and compound forms not infrequently occur, concerning which equally able pathologists might take different views. And it is necessary to insist that in different parts of the same tumour considerable variation in structure and arrangement occurs. Thus at one part there may be a typical

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sarcomatous appearance, while at another region of the same growth glandular-like features may be apparent. With this word of caution we may now proceed to consider the various forms of renal tumour.

True Renal Tumours.

I. *Tumours of Connective-tissue Type.*—These we can divide into the clinically convenient groups: (1) simple and benignant, and (2) malignant or sarcomatous. The simple or benign growths are exceedingly rare and of but little practical importance. Fibroma, lipoma, and angiomatous formations are occasionally met with. The fibroma usually occurs as a small rounded nodule; occasionally they may be multiple. True lipoma is exceedingly rare; some so designated have probably been growths arising in aberrant adrenal tissue. Fibro-cystic and fibro-lipomatous growths have been removed by operation, but it is probable that some of these have been perirenal in origin. Angiomata are occasionally met with, and closely resemble similar formations met with in the liver.

At the present time most renal tumours, especially those occurring in early life, are considered as belonging to the malignant connective-tissue type. Renal sarcomata have been conveniently grouped according to the period of life at which the tumour develops: (1) fœtal sarcoma; (2) sarcoma of infancy and childhood; (3) sarcoma of adults. They have also been classified according to their seat of origin. This is a grouping of considerable pathological and clinical importance.

1. *Capsular Sarcoma.*—The tumour originates in the renal capsule. The kidney is displaced, distorted, or invaded, and sometimes may be actually surrounded by growth.

2. *Hilum Sarcoma.*—Here the starting-point is the connective tissue of the pelvis and calyces. The growth is at first outside the kidney substance. Later the organ becomes invaded, expanded, and flattened out.

3. *Perirenal Sarcoma*, as an extra renal tumour, should, strictly speaking, not be included here, but at all events such cannot at present be distinguished clinically from true renal growths, and hence it is well to mention them. They develop in the fibro-lipomatous tissue immediately surrounding the kidney, and although consisting often of fibrous and fatty elements, generally contain sarcomatous cells.

Histological Arrangement.—At the present time a study of histological features offers the most interesting and fruitful method of grouping, and is likely to remain so, for a basis dependent on embryological considerations offers peculiar difficulties and opens a way to almost insuperable perplexities.

The greater number of the sarcomata consist of *round-* and *spindle*-cells. The *striated* cell met with in the so-called myosarcoma is frequent in congenital growths. A vascular form, or *angio-sarcoma* occasionally occurs.

Glandular-like structures not infrequently are met with, combined with undoubted sarcomatous cells, and such a growth is sometimes termed an *adeno-sarcoma*. Compound and complex forms also occur. Melanotic sarcoma is always secondary.

II. *Tumours of Epithelial Type.*—Here it is somewhat more difficult to sharply distinguish between benign and malignant forms. In this class we place—

1. *Adenomatous growths.*—Simple adenoma may occur either single or multiple. Solid and cystic forms are recognised. The trabecular cystoma may perhaps be justifiably placed in this group. It occurs as an aggregation of minute cysts, each lined with cubical or columnar epithelium. The papilliferous cystoma usually presents malignant characters. Its chief features you will be able to see in the sections I have placed under the microscopes.

2. *Carcinoma.*—Carcinoma of the kidney is apparently rarer than was formerly believed when almost every renal tumour was labelled "cancer." Knowledge based on recent investigations is unfortunately meagre. Renal carcinoma seems hardly ever to occur in early life. It is rare before middle age, but after fifty increases in frequency. Two forms are recognised, the glandular, or true renal carcinoma, which may assume a nodular or infiltrating form, arising in connection with the epithelial elements of the renal parenchyma; and the papilloma, which occurs in the pelvis of the kidney, and in structure closely resembles the vascular villous growths commonly met with in the bladder. I believe the renal papillomata are almost always malignant. They usually give rise to profuse hæmorrhage and often cause renal colic, and some degree of hydronephrosis may occur. Such growths have been dealt with surgically.

In my next lecture I hope to refer more particularly to growths arising during intra-uterine development and those met with in young subjects.

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AN ADDRESS

ON

SOME AFFECTIONS OF THE CONJUNCTIVA.

Delivered before the Wimbledon and District Medical Society.

By A. QUARRY SILCOCK, M.D., B.S.Lond., F.R.C.S.,

Surgeon to St. Mary's Hospital, Paddington, and the Royal London Ophthalmic Hospital.

MR. PRESIDENT AND GENTLEMEN,—In the first place I have to express my appreciation of the compliment which you have paid me in asking me to come here to-night. It gives me the greater pleasure to do so inasmuch as your President, Mr. Collins, is a very old friend and fellow-student of my own.

I suggested to Dr. Deas that I should give an address on a treatment of some of the commoner forms of eye affections, because I thought it would excite a discussion, which might be the more valuable in that we are apt to neglect to investigate and to reflect upon the causes, the consequences, and the treatment of diseases we regard as being of rather minor import, or may be to ignore them. Thus slight degrees of inflammation of the conjunctiva are often overlooked; sometimes they are often vigorously and even aggressively treated, yet perhaps to no avail, the cause being unsought. Yet a little conjunctivitis is often sufficient to spoil an operation for cataract, and may indeed lead to corneal ulceration and other like troubles.

One of the commonest forms of conjunctival disease is called conjunctival catarrh. Now what is conjunctival catarrh? It is the resultant of hyperæmia of the conjunctiva caused by varied forms of irritation; by mechanical forms, such as the presence of dust, or by certain chemicals, or very commonly indeed by micro-organisms. Such hyperæmia may be excited by reflex disturbances induced by the errors of refraction, hypermetropia

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in particular. As a result of the hyperæmia there is at some time or other an increase in the mucous secretion of the conjunctiva and an increased exfoliation of the epithelium cells. Thus a quantity of sticky mucus accumulates about the conjunctival surfaces, leading to a gumming together of the edges of the lids and to a sensation of grittiness as of a foreign body being in the eye, a symptom people affected so often complain of. Sometimes they will tell you that they feel unable to lift the upper eyelid, especially after having been to sleep. The reason, I take it, is that the palpebral and ocular conjunctival surfaces become a little sticky, and there is in consequence a real momentary difficulty in raising the lid. Catarrhal conjunctivitis, like catarrah of other mucous membranes, is much more common in advanced age. It is also common in children, who are more liable to infect their eyes by rubbing the lids with dirty hands than are young adults or people of middle age. The treatment of this condition is very simple. In the first place, it is absolutely necessary to find out what the cause may be. Never omit to evert the upper lid and look for a foreign body. It is a common experience to see people who have been treated for catarrhal conjunctivitis due to a small foreign body slightly embedded in the conjunctiva of the upper lid or cornea which has been overlooked. Again, it is often set up by some obstruction to the outflow of tears: press upon the lacrymal sac and canaliculus with the tip of the forefinger, and if there be any obstruction an immediate regurgitation of the contents of the sac into the canaliculus will take place, and you will see it flowing out of the punctum into the conjunctival sac. If there be any error of refraction, let it be corrected by proper lenses; foreign bodies must, of course, be removed and lacrymal obstruction dealt with if present. I believe the most usual local treatment is to drop in sulphate of zinc and nitrate of silver solutions or some other irritating astringent drug. What is the result? That there is immediately an increase in the hyperæmia and a flow of tears, and I think it is this flow of tears which plays a most important part in the remedy. The flow of tears is the natural means of irrigating the conjunctiva. Tears are constantly flowing into the conjunctival sac and out again, and so the surface of the mucous membrane is kept comparatively free from micro-organisms, and it is this natural method of irrigation which may be imitated in the treatment of conjunctival inflammations with advantage. Where you have to do with a little catarrh all that is necessary is to wash away the sticky mucus; and that you can do, as in the case of the throat, by using a physiological solution of salt and water. If you like you can use a solution of bicarbonate of soda. If you think that the conjunctival inflammation is due to micro-organisms, as it so often is, then you may reasonably employ a weak antiseptic solution, say perchloride of mercury 1 in 5000 to 1 in 10,000, or weaker still, or, in the same strength of solution, biniodide of mercury. Or you may order that universal remedy boracic acid in saturated solution, which is not so strongly germicidal as the mercurial solutions. But the way in which the conjunctiva should be washed out is a matter of some moment. It is practically futile using an eye-glass in the ordinary way: the eye-glass filled with fluid is tilted over the eye so that the upper lid is thus pressed down on the surface of the globe, consequently none of the fluid gets into the upper fornix of the conjunctiva. What you should do is to lift up the upper lid and wash out the upper part of the conjunctival sac with the solution as well as the lower. I have often thus succeeded in rapidly curing infective forms of conjunctivitis which had been treated for days by simply bathing the eye with boric acid lotion, or the putting in of drops of sulphate of zinc. The washing out is best done with the aid of an irrigator. I have brought down nozzles which we use at Moorfields. They are of glass, so that they can be easily sterilized, and are attached to the tube of the irrigator; they, as you see, can readily be inserted beneath the upper lid. I have also brought one of my own, which, being flatter at the lip, is more easily placed between the upper lid and the globe. In using these, it is of the utmost importance that the cornea should not be damaged by them, for if you produce a little abrasion infection may follow and a troublesome ulcer result; organisms can readily make their way into the corneal substance if that natural barrier against infection, the phagocytic epithelium, is scraped off.

It is a very simple matter to prevent the gumming or sticking of the edges of the lids together by smearing upon them a little antiseptic ointment, or if you have not that, some vaseline.

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Passing on to other affections of the conjunctiva, one which is frequently met with is the so-called phlyctenular conjunctivitis. The phlyctenule consists of a little injected nodule, or there may be several of them, beneath the conjunctival epithelium, superficial to the sclera, from or to which run a few dilated vessels. They often give rise to comparatively little pain and trouble. If they are situated at the margin of the cornea, however, they may cause considerable irritation, and if they are in the cornea itself, to very great irritation. What are these phlyctenules? If you excise one and examine it with the microscope you will find that they consist of epithelioid cells and leucocytes, which are often enough engaged in a lymphatic space. I take it that the phlyctenules arise in this way:—The infective material, organisms, or other toxins invade the subconjunctival lymphatics; it is arrested at one point, and there leucocytes collect. The epithelioid cells of the lymphatics aid the latter, and you have a clump of cells resisting its progress more or less effectually. If the infection has the better of the contest the nodules break down, together with the overlying conjunctival epithelium. We then have a phlyctenular ulcer. Phlyctenules and phlyctenular ulcers are very frequently met with around the margins of the cornea. Why is that? You will remember that a number of the subconjunctival lymphatics end in cæcal extremities at the margin of the cornea; in these the virus is filtered off, as it were, begetting an accumulation of phagocytic cells. Some of the subconjunctival lymphatics, on the other hand, communicate with the corneal interspaces; thus infection may reach the cornea and give rise to corneal ulceration. The simplest treatment in such cases is to wash out the conjunctival sac with a weak mercurial solution, or with some other weak antiseptic, boric acid for example. If the cornea is affected on the phlyctenules at its margin, you may put some atropine into the eye. Atropine, however, should be used with discretion. Atropine has no sort of direct action on the inflammatory lesion, so far as I know. One uses the atropine as a mydriatic, or in order to give the ciliary muscle physiological rest, and therefore no more than is necessary for the purpose should be employed. If strong solutions of atropine are instilled, disagreeable consequences may ensue; a rash like that of scarlet fever may appear, with a high temperature. In the case of a boy æt. 6 I once dropped into both conjunctival sacs a drop of a 2 per cent. solution of homatropine one evening about five o'clock, to examine his eye. He was delirious the whole night, and had a temperature of 104°; he had absolutely no sleep. Next morning he had recovered himself to a certain extent. But as night approached he again became delirious, and it was not until the early morning of the second night that he slept at all. Such symptoms are very alarming to patients. So in a child half a grain or one grain solution of atropine to the ounce is sufficiently potent under ordinary circumstances, used twice or thrice a day.

In my opinion, the conjunctival infection may reach the ciliary body by the subconjunctival lymphatics. I believe that certain forms of cyclitis, and even iritis, are the result of the absorption of either toxins or micro-organisms from the conjunctival sac. Occasionally, after an attack of acute conjunctivitis, you find the patient complaining of intense pain in the eyes. If you examine such an eye you may find there is much ciliary congestion and great ciliary tenderness, so much so that it is difficult to palpate the eyes and determine the state of the intra-ocular tension without giving great pain. A gentleman who consulted me said that on a certain day he had found that the right eye was becoming painful, or at all events it was in a condition of some discomfort, from the fact that, as he said, there was "a grittiness in it." He thought he had something in it; in fact, he went to a surgeon to ask him to see if there was. I saw him the next day; he had had no sleep during the night by reason of pain; the subconjunctival tissues and ciliary region were deeply injected, the ciliary region intensely tender; the pupil was of the same size as that of the sound eye, and its reflexes were good. Complete rest at home was ordered; boric acid fomentations were used, a little boric acid ointment to put on the edges of the lids to prevent them gumming together, and in a week he was quite well. In such a case it is sometimes anything but easy to make a diagnosis. If you use a mydriatic, such as a weak solution of atropine, you will not find any synechiæ nor evidence of exudation from the iris or ciliary region, and the media are quite clear. I have known cases of the kind to be called and treated as glaucoma, and it is not difficult to understand

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how such an error in diagnosis might happen. If you use eserine you probably make the condition worse; moreover, to put in eserine into a very congested eye is to increase that congestion, and consequently the pain; moreover, if iritis should supervene, a contracted pupil is very undesirable. It is a matter of conjecture on my part that the ciliary region is involved by absorption of conjunctival toxins. I have no scientific proof of this proposition, but there are certain pathological evidences which bear the theory out. I do not think that the organisms get through in all cases into the ciliary region; more commonly probably it is their toxins. It is possible to prepare from the *Staphylococcus pyogenes aureus* a crystalline material which, if put into a rabbit's eye, at once gives rise to diffuse keratitis.* So there is nothing out of the way in supposing that toxins absorbed into the ciliary region may induce such a condition as I have suggested.

Now I come to another important class of cases which you may meet with in general practice, and that is purulent conjunctivitis or ophthalmia. Purulent ophthalmia is always the result of infection by micro-organisms. In the new-born infant it is practically invariably due to the gonococcus, and if untreated the results, as I dare say you know, are absolutely disastrous. It you have any reason to suppose that the mother of the child is infected with gonorrhœa, the right thing is, before delivery, to wash out the vagina as thoroughly as you can, and the moment the child is free of the mother the face should be washed, the eyelids being carefully sponged over with perchloride or biniodide of mercury solution. When the eyelids are opened a drop of nitrate of silver solution (2 per cent.) is put into the eye. This is the famous Credé treatment, as it is carried out in Leipzig. Since that treatment was adopted there the number of cases of lost eyes as a result of ophthalmia neonatorum have diminished enormously. But even though you do this it is not a prophylactic; the disease may go on, and the question is, what is the best way of dealing with such a case. Personally, here, also, I think the right thing to do is to wash out the conjunctiva with antiseptic solution, for example, biniodide or perchloride of mercury of a strength of 1 in 10,000 or 1 in 5000 in the way which I have indicated. This should be done constantly. It is not enough to do it two or three times a day only; it must be done a dozen times a day, and the child must be awakened to have it done, and when it is not being done a moist antiseptic compress should be kept on the eyelids, with some ointment between the edges of the lids to prevent their sticking together. The outer canthi may be divided, in order the more readily to evert the lids and wash out the fornices, if necessary.

The solution of perchloride of mercury may alternate with physiological solutions of salt or of saturated solutions of boric acid. But the utmost precaution must be taken never to scratch the cornea. A touch on the cornea with one of the irrigator nozzles would produce an abrasion, and there would follow inevitably an ulcer, which would probably lead to panophthalmitis. I take it that if the gonococcus gets inside the eye it causes suppuration where it affects a lodgement. You have to depend entirely on the natural resistance of the tissue-cells to prevent the organisms from reaching the interior, and if you use strong solutions of nitrate of silver, say twenty grains to the ounce, I think you very often do more harm than good, because you diminish or destroy this, and you only kill the organisms which happen to be on the surface of the conjunctiva, and those you could have got rid of by simply mechanical washing without injury to the tissues. Another objection to the use of nitrate of silver is that it may not only fail to cure the disease, but if it is used too often or in too strong a solution it may induce some sloughing of the eyelids. I have come across cases in which strong nitrate of silver solution dropped into an infant's eyes three times a day resulted in considerable deformity. So I wish strongly to recommend the line of treatment I have mentioned, and I think the Credé treatment should be carried out.

* Mr. H. G. Plimmer has given me the following references:—Leber ('Fortschritte der Medicin,' vol. vi, 1888) isolated a crystalline substance from *Staphylococcus pyogenes aureus*, with which he produced "une inflammation dans l'œil du lapin;" and Christmas ('Arch. de l'Institut Pasteur,' 1888) isolated a substance which produced "un œdème de la conjunctive et une suppuration legère dans la chambre antérieure de l'œil du lapin." He also found the dead staphylococcus injected into the anterior chamber produced "l'œdème de la conjunctive, la décoloration de l'iris, et la formation de pus dans la chambre antérieure."

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I should like to allude now to diphtheritic ophthalmia. I have already said a membrane may be produced by certain chemicals, and it may also be produced by organisms, undoubtedly by the staphylococcic and streptococcic forms, as well as by the diphtheria bacillus. Only occasionally do you meet with diphtheritic conjunctivitis. It is of very great importance, and sometimes difficult to diagnose. I will read the notes of a case of this kind, if you will allow me. A boy æt. 4 was admitted as an in-patient to the Moorfields Hospital on May 8th. He was very ill, had great pallor and puffiness of the face and extreme œdema of the forehead and over the left temporal fossa. In the centre of the forehead was a sore covered with membrane, and the lids were blown out and membranous. On removal of this membrane there was left a raw bleeding surface. On the cutaneous surface of the lower lid, where it was overlapped by the upper, was a patch of membrane covering a raw surface. There was much muco-purulent discharge. The right cornea was clear, but the left showed an extensive superficial ulcer. There was nothing abnormal in the throat except enlargement of the tonsils. We took cultivations of the conjunctival exudation, but we could not demonstrate the Klebs-Loeffler bacillus. The temperature was slightly raised; the pulse was very rapid. The patient was given ether, and while under its influence the membrane was removed from the lids, and both conjunctival sacs were irrigated with a solution of perchloride of mercury 1 in 4000. This irrigation was repeated every hour, the membrane re-forming only to a slight extent. However, the condition improved so much that we doubted whether it was true diphtheria until the characteristic nervous symptoms manifested themselves. On June 2nd the child was observed to be suffering from weakness and unsteadiness of the lower limbs, and the knee-jerks were absent. There was no wasting of muscles, but they were flabby. On the 5th there was extreme weakness of the upper extremities, and on June 17th the child developed well-marked right facial palsy. At no time was there difficulty in swallowing or regurgitation of fluid through the nose; but the pulse was very rapid, varying from 120 to 140. The temperature was never above 105°. On June 15th the child was extremely ill, the pulse was very rapid, and the child was so weak that we did not think he had any chance of recovery. He was fed with great care, and from that time he began to improve. Subsequently, when I saw the boy he had recovered free use of his muscles; there was some corneal opacity in the left eye, and still some ciliary injection. The right cornea was perfectly clear, and there was nothing abnormal about the external appearances of the eye. Here, again, the treatment was of the simplest possible character, simply irrigation with, I think, an unqualified good result. The serum treatment was not used, for the case was far advanced when it was first seen, and as it rapidly improved we did not consider the antitoxin to be indicated.

A

DEMONSTRATION TO POST-GRADUATES

IN THE

OPERATING THEATRE OF WESTMINSTER HOSPITAL

ON

ANTISEPTIC METHODS AND APPARATUS.

JUNE 10TH, 1901.

By WALTER G. SPENCER, M.B., M.S., F.R.C.S.

GENTLEMEN,—Antiseptic methods, as you are well aware, were first introduced by Lord Lister, and with certain modifications, especially those which he later on himself adopted, hold the ground to-day. I thought it would be profitable to consider this matter first in our demonstration. And then, secondly, we might pass on to aseptic measures, those by boiling and the application of steam. But these latter, except in a limited way, entail more or less expensive apparatus, and consequently are not so well fitted for private work as for hospitals.

With regard to antiseptics, the first substance I shall mention is undiluted carbolic acid, a body which has anæsthetic properties, so that if we are scrubbing out an inflamed wound with it the patient will not need to have opium as he would in former days, when the substance used was nitric acid. But after it has been used in that way the wound must be thoroughly washed, so as to clear

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away any remaining quantity of the acid, either with distilled water or with absolute alcohol. If you have a narrow sinus, and you are going to put carbolic acid into it, you should wash the skin around first of all with the absolute alcohol, and then you find that should any of the carbolic overflow it does not blister the skin. Next we have carbolic acid in a solution of 1 in 20, or 5 per cent., which forms the stock solution. It is specially employed for the submersion of instruments in trays before use. The chief point in regard to it for that purpose, as compared, for instance, with such an antiseptic as perchloride of mercury, is that it does not tarnish nor destroy the instruments. But it has this objection, that it will shrivel up the hands of the surgeon. The general custom is to pour into the instrument tray the 5 per cent. carbolic acid, and then at the commencement of the operation dilute down to 2½ per cent. This carbolic acid, 1 in 20, is also used for scrubbing out infected wounds of a larger character than those for which we can safely employ undiluted carbolic, it should likewise be washed away with water afterwards. Carbolic acid, 1 in 40, is not so valuable for the immediate destruction of germs, but it is strong enough for the purpose of swilling the hands of the surgeon and rinsing sponges during the operation—I shall come back to consider the hands of the surgeon by and by—in such a lavabo as I show you here, in which one can constantly wash one's hands as they become soiled. But we do not now use carbolic acid for purposes for which it was employed in the first days of antiseptic surgery, not for the peritoneal cavity; we do not inject it into large abscess cavities, such as a psoas abscess. The broad principle is that you never inject this strong antiseptic, and the same remark applies to perchloride, unless you are certain you can wash it out again. Therefore, that precludes the absorption of carbolic acid from such large cavities as the peritoneum. As a matter of fact, employing carbolic acid in the limited way I have described, we do not see carboluria or coma resulting, as was formerly the case.

Next we will pass to perchloride of mercury. We have in this substance a very valuable antiseptic. There are some who prefer the iodide, it is a little less irritating and not so strong; there are some who use the corresponding cyanide. But the perchloride is the strongest of the three, and the characteristics much the same, except that the others are less irritating. Perchloride of mercury can be specially thought of as being used for the skin, but not for instruments or for metal washing bowls. The metal of bowls or instruments is quickly destroyed by the mercury, being eaten through by it. On the other hand, the metal bowls and instruments destroy the strength of the solution by precipitating the mercury, so that either way perchloride of mercury will not do to come into contact with metal. But it is very suitable for the skin of the patient, and also for the hands of the surgeon. For the hands, and for the skin of an infective case, we may use perchloride, 1 in 500. Here I show you an alcoholic solution of it, which is rather strong. Some prefer to add soap, soft soap in solution with 1 in 500 of the perchloride, which is the alcoholic mercurial soap which you see here. What makes perchloride of mercury so convenient is that it can be carried about in blue tabloids, one of which is dissolved into one or two pints of water; there are also iodide tabloids, stained brown, making a strength of 1 in 1000, or 1 in 2000. It also serves the purpose of swilling the hands during an operation. But, again, we do not employ perchloride of mercury in the same way as it was formerly used, that is to say, in the peritoneal cavity or for large abscess cavities. Of course, we should certainly never employ it for the regions of the mouth. Even in places where perchloride of mercury is less liable to cause trouble, such as in the vagina, it should be got rid of by the use of water afterwards. If perchloride goes up into the uterine cavity, say after labour, and remains there, the patient being susceptible to mercury, it may prove fatal to her, as has happened in many cases. This substance should, indeed, not be used in the female organs by means of the douche, but only by swabbing through a speculum, using pure water swabs afterwards to get rid of the perchloride.

Moreover, we have antiseptic powders; one of the chief ones is boric acid. The great value of boric acid in surgery is that it is the best substance for fomentations, fomentations of thick boric lint; or you can simply take a piece of lint, put it into a bowl, sprinkle plenty of this powder over it, cover with boiling hot water, and then turn it out dry. This makes a proper surgical poultice, and it is

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much preferable in this connection to carbolic acid fomentations. A carbolic acid fomentation is never good; for one reason, if you have a fomentation over a large skin surface there is likely to be absorption. It has been used for whitlows of the fingers, since it has some anæsthetic property and so relieves the pain; but it has been a cause of gangrene of the fingers. Cases of that kind are being continually recorded—I could quote several—where the use of carbolic acid has led to gangrene. For broken skin or wounds boric acid fomentations are a good application, and as such are fairly antiseptic if changed about every two hours. The antiseptic strength of boric acid is not great, and decomposition will ensue if you leave it on long; but as the fomentation will certainly get cold in two hours, that should be the limit of time during which it is kept on. We use boric acid as a dusting powder in addition, or as a sort of backing, to an antiseptic dressing; but it is not so important as the next one I am coming to, namely, iodoform. Boric acid as a lotion is of very little antiseptic power. It has this practical good, however, that supposing you have some water which you boil, but owing to certain exigencies you are compelled to keep standing for several hours or overnight, it is better to add to it boric acid to prevent a certain amount of growth of germs which would otherwise occur in the meantime.

A powder which we are constantly employing in surgery is iodoform, the great value of which is that it is a less soluble material, and therefore gives off its antiseptic property, iodine, extremely slowly and yet continuously. And owing to its chemical formula being related to a certain extent to chloroform—the formula for chloroform being $CHCl_3$, and iodoform CHI_3,—it has a certain amount of anæsthetic property when applied to a painful wound or surface. It is of great service, therefore, in all wounds which are being treated by second intention, to prevent the decomposition which might otherwise go on in connection with them. Especially that applies to wounds or sores in the mouth or the rectum. It is generally employed along with gauze, either this iodoform ribbon gauze for small sinuses, or as these strips of gauze which you see rolled up. There is no doubt about the value of gauze plugging, which takes up the discharges from a granulating surface or an abscess cavity. This iodoform gauze has revolutionised to a large extent the treatment of septic inflammation. In former days septic inflammation was treated by leaving it as long as possible, and then making a small opening and letting out the pus. The result was a long-continued septic absorption, and at the end a sinus to be cured. Then came the stage of drainage-tubes, when the abscess cavity was opened a little more freely, sometimes by more than one opening, and drainage-tubes inserted. The patient gained this benefit, that he did not have septic absorption, but he had still a sinus along the course of the drainage-tube to heal up, which caused delay, in some cases complicated by tubercle. The tendency now is to lay an abscess cavity as widely open as possible, so that it is practically reduced to a hollow ulcer, and dress it daily or every other day with iodoform gauze. The result is that the patient is immediately saved from septic absorption, and, secondly, he escapes from any trouble about sinuses, for the ulcer heals up quickly from the bottom. We use iodoform not only as a powder, but also as an emulsion, either with glycerine as you see it here, or some people prefer it in a saturated solution of alcohol and ether. As such it is especially fitted for injecting tuberculous cavities, joints, and other collections, such as psoas abscess. In some of the cases, where the tubercle lies on the surface of the cavity, when the iodoform can come in contact with the tubercle bacilli, it has a very beneficial effect. It fails when the tuberculous process infiltrates far beyond the wall of the cavity or the joint. But you are thinking about the objections in regard to iodoform. First of all as to its smell. I do not think we need say very much about that, except that it constitutes an objection in private houses. Otherwise, if it is necessary to use it, we find people get accustomed to it. It is not much good attempting to cover up the smell, or to substitute any other products which are said to replace iodoform, because by so much as they get rid of the powerful odour of the iodoform, by so much do they become useless for the special purpose.

With regard to iodoform poisoning, we do not see iodoform poisoning, and I am very sceptical about it. Patients after the use of iodoform have suffered from drowsiness, or even coma, on the one hand, and, secondly, they have suffered from trouble of the kidney, especially hæmatinuria. But

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I believe it will be found that some of those cases were due either to impure iodoform on the one hand, or to it being used at the same time as some other strong antiseptic which irritated the kidney. One can imagine that if you use a strong antiseptic and irritate the kidney it will not excrete so readily, and there will be a retention of iodoform in the system, which may induce the comatose condition. We try to avoid such accidents by keeping our iodoform in crystals, and whenever we use emulsion we take care to have it freshly made, for after about three weeks the emulsion begins to decompose; and moreover, as a matter of fact, we do not now use the strong antiseptics which will damage the kidney. If you use iodoform freely you can test the presence of iodine either in the patient's urine or in his saliva. Yet iodoform has been given in the form of a pill for the cure of phthisis until the patient has reeked of iodoform, and could taste nothing but iodoform in his food, and yet he has suffered from no symptoms except from some drowsiness.

There have been other proposals with regard to antiseptic powders; also there has been a paste suggested, which may be of some service for first aid in this country; in South Africa it was very serviceable. It was proposed by Mr. Cheatle. The paste is carried in a collapsible tube, and is intended to be spread over wounded surfaces immediately, without attempting to clean the wound first of all, leaving that until the patient can be brought into proper surgical conditions. This paste consists of mercury and zinc cyanide 400, carbolic acid 40, tragacanth 1, and water to 800. You can easily understand it would be useful in war, for on going to the patient you can put some of this paste on without touching the wound, but leave it to be cleaned up later under favourable surgical conditions.

In the antiseptic gauzes which we use we also follow on the lines of Lord Lister. We employ the pink gauze impregnated with the double cyanide of mercury and zinc, for the reason that whilst it is truly antiseptic it is at the same time fairly non-irritating if it is made as he ordered it to be made. But if the manufacturer tries to scamp his work and leaves some free perchloride of mercury in it, that free substance will irritate in some cases. This original perchloride of mercury gauze is stained with aniline blue, and is a good gauze as far as its antiseptic properties are concerned, but in a person who sweats freely and whose discharges are irritating, and especially in hot weather, this gauze will very likely raise a pustular eruption of a troublesome nature, which may take some time to heal. Therefore, now this sal-alembroth gauze is not so much used. When we want a non-irritating gauze we choose thymol gauze, and in that case we do not expect it to have very strong antiseptic properties. On the other hand, the wool we are constantly employing, as the cheapest and best kind of wool, is certainly this blue perchloride of mercury wool. Sometimes, in hot weather and in a sweating patient, it causes iritation, then we fall back upon iodoform gauze and wool. But the great objection to iodoform gauze and wool, from a hospital point of view, is its expense. Whereas perchloride of mercury wool is about one penny per pound more expensive than ordinary white wool, iodoform gauze, if properly made—I believe some manufacturers cheat by substituting chinosol and creolin,—is expensive, and for private cases there is the disagreeable smell.

A good deal of comfort to the patient, and more than people think, rests on the sort of bandages which one employs. There are cotton bandages here which are employed because they are cheaper than the others, but are inelastic, when they are bound tightly. These other bandages of domet flannel I show you are much more comfortable to the patient. If you buy them at the chemists you have to pay a ridiculous price for them, but if you buy the flannel from a draper's in strips more than two feet broad you can get it for about sevenpence a yard. The result is the bandages come as cheap, if you make them in that way, as do cotton bandages. Some people have proposed to use perforated rubber bandages, such as those employed for ulcerated legs, but the patients do not like them, they feel very much tightened up and oppressed by them.

Such is a short sketch of some of the antiseptic materials used.

We will now turn to asceptic matters. With regard to sterilising, it entails apparatus, which on the whole is expensive and troublesome unless surgery is carried on on a large scale. I show you a boiling apparatus. Here is the simplest form of apparatus which you can have for boiling. It is filled with a soda solution, which is made by

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adding a teaspoonful of bicarbonate of soda to a pint of water. It has a tray on the principle of a fish kettle, and can be used for boiling either instruments or towels and dressings. You can use dressings, such as butter-cloth or muslin, and towels, or lint and boil them in the way you see, but the objection to anything of that sort is that the material comes out of the apparatus wet. So that when we want to sterilise dressings and to bring them out fairly dry it is necessary to have a steam vessel. Here is a steam arrangement of a simple character, enclosed with a jacket, and with an escape for the waste steam. Inside there is generated steam under pressure. There the dressings are packed, and are kept heated for half an hour or an hour. At the end of that time it is necessary first of all to let the steam off, before the top is removed, so that at the end the dressings come out fairly dry. Of course care must be taken to see that the kettles are packed in the inverse order to that in which the contents will be required, bandages which are wanted last being at the bottom, otherwise there will be confusion, and the whole lot must be turned out before you find what you want. The holes at the side can be opened, and then the steam penetrates to every part of the dressings. Afterwards, by turning the slide the holes are closed and the kettle becomes shut off. This apparatus which I have shown you is neither the largest nor the smallest size. There are certain advantages which may be named in connection with this method of sterilising. If a towel is wetted with carbolic acid and placed in contact with the skin it may cause irritation, whereas the towel taken from the sterilising apparatus such as this is dry and does not irritate. There are elaborate methods of applying dry heat, but dry heat requires even more expensive apparatus than moist heat.

We will now pass on to consider some other things.

First of all with regard to the sponges we employ. The ordinary sponges we make consist of cotton wool twisted up in a square of gauze, which are then sterilised. I show you how they come out of the steriliser. Wool has to be heated first of all to be expanded before it is screwed up into these little dabs. They serve as wipers, and, to a certain extent, are absorbent, and are afterwards thrown away. There are some surgeons who use them entirely. Some simply cut out squares of lint, pack them in a pile and sterilise them, and simply depend upon them for wiping. But for certain purposes, at any rate, I think nothing can supplant the marine sponge, either in the form of button sponges or as flat sponges. Many surgeons, however, feel so uncertain about them that they do not use them. We use them here, and I employ them also in private. I am certain that they can be effectively sterilised, and that they are certainly free from any danger of infection. The way to prepare a marine sponge for surgical use is to first of all get rid of as much sand as you can by shaking it or beating it in a bag, then knead it in water until the water comes away from it clear. In that way the marine matter is largely removed from it. Next the sponge should be put into an acid, one which will destroy the germs and will not destroy the sponge. The acid which we generally employ for the purpose is sulphurous acid, 20 per cent., or 1 in 5. Other people prefer hydrochloric acid, not too strong, 8 per cent. is quite strong enough. The sponges are left in that acid for as long as twenty-four hours, and will not hurt if they stay even longer, and are then put into carbolic acid of 5 per cent. strength, where they are kept until they are wanted. We generally keep them a week under carbolic acid before use again, as the carbolic acid does them no harm, except to make them a little brown. When we put them afterwards through the sulphurous acid this again whitens them. Some people think—though I do not consider it necessary—that additional safety is obtained by putting the sponges through permanganate of potash, 1 in 400, for a day, until they are brown, and then removing the brown colour by a saturated solution of oxalic acid. Some say, "Cannot you boil them?" As a matter of fact we have tried boiling sponges. The substance to boil them in is tannate of potash, consisting of potassium hydrate 1, tannic acid 2, and water to 100 parts. That substance will not alter the texture of the sponges, even when they are boiled long enough to destroy all germs in them. But if one uses a marine sponge for the purpose of clearing away some very foul material, the safest way is to burn it.

Now I come to speak of the preparation of the skin of the patient and the hands of the surgeon. About this subject there has, of course, been, and

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there still is, a good deal of controversy. I can only say that the statements I am making are founded upon good evidence, both practical and otherwise. The skin of the patient requires first of all to be obviously clean, and that is the best point to start from. We often have patients in to whom a good deal has to be done before the skin can be called even obviously clean. In some cases the skin has to be almost fomented before it can be termed obviously clean. Let us start, then, at the point where the skin looks clean. Then it has to be shaved, and, in most cases, especially in patients with fatty, greasy skins, you must get rid of this fat by one or all of these three materials: methylated spirit, ether, or turpentine. You have now got the skin obviously clean, you have got rid of the hair, and you have removed the fat. Then you require to wash the skin over, either with carbolic acid, 1 in 20 or 1 in 40, if it is very thin, or with perchloride of mercury, 1 in 1000 or 1 in 2000. By that means you have a skin which is, surgically, fairly clean, as differing from the previous stage of obvious cleanliness. In most cases, after such a preparation, you can proceed to do an operation without any danger. But in certain places, especially where the skin is thick, it is wise to apply for some time before the operation what is practically a carbolic acid fomentation. It is called a carbolic guard or carbolic compress of lint squeezed out in 1 in 20 carbolic, and applied to the part which has been made surgically clean. This must be applied not more than two hours before the operation, even in the case of thick skin. All the service it will do in the way of killing germs a little beneath the surface, in the mouths of the hair follicles and sebaceous glands, will have been done by that time. In susceptible patients, if the guard is kept on longer than that there may be carboluria. In children, it must not be applied for more than one hour. Mistakes have been made, for it has happened in hospitals that a child has been prepared perhaps several hours beforehand, and carboluria and coma have supervened. There are also places where a carbolic acid compress is entirely unsuitable. That is especially the case for fingers and toes of old patients; there is ever a danger of senile gangrene, for the constriction of the arteries set up by the carbolic may be enough to turn the scale in the direction of gangrene. Nor should the compress be applied to the face, because that part is very easily cleaned, is plentifully supplied with blood, and heals readily. Moreover, if the compress were used there, there would be danger of the carbolic acid getting into the mouth and eyelids. If you want to apply a guard to the face, it should be one of boric acid and not of carbolic acid. In any case of a person who has disease of the kidneys, such as albuminuria, it is well to err on the safe side, and not to use a carbolic guard anywhere, but a boric acid one. Never use a perchloride guard, if you do you are liable to set up a most severe pustular eruption, which will be very troublesome to heal.

As to the surgeon, let us again start from the point of obvious cleanliness, the nails also having been carefully prepared and cleaned. Here, again, in connection with this part of the subject there has been a very long controversy as to how you can best clean the hands of the surgeon. Investigators on the subject have produced confusion, either by experimentally soiling the hands with some very virulent material first of all, or else they set up this limit, that we must make the hands bacteriologically clean and germ-free. Now, in the first place, the surgeon starts from the point that his hands are obviously clean, and not that of hands infected by some virulent material. And, secondly, I think it may be said that to obtain hands absolutely free from germs is beyond possibility without destroying the skin itself. What the surgeon wants is to have his hands free from germs which will harm the patient, and that is obtained by starting with the hands obviously clean, the nails pared, and then washing them thoroughly with soap and water, afterwards rinsing them in 1 in 40 carbolic acid, or 1 in 2000 perchloride of mercury, or the corresponding iodide. If that is done I am sure no harm will happen to the patient from anything which may get into a wound from the surgeon's hands. If during the operation he does not soil them with virulent stuff, and if he swills them frequently in either of those two solutions which I have mentioned, I believe his hands will be perfectly safe. Some propose much more severe measures, which will not improve the hands. Alcoholic soap, with perchloride of mercury 1 in 500, is a very valuable thing if your hands have been previously soiled by some dangerously infective material, such as from erysipelas or puerperal fever, and then, no doubt, it is

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an extra precaution which is required. You should take a piece of gauze which has been dipped in perchloride solution, or smeared with the perchloride soap, and rub the hands well. Some think that the only satisfactory way is to soak the hands until they go brown in this permanganate of potash 1 in 400, and having done that, plunge them into a hot saturated solution of oxalic acid for another five minutes to get rid of the brown colouration. Measures which tend to excess are always liable to be followed by reaction in the direction of too little precaution. One would prefer to keep to the happy mean. I do not think such courses are generally necessary, although they are proper when the hands have been infected by some virulent material and another operation has to be performed shortly afterwards. Some surgeons maintain that gloves should be worn during an operation. Two forms of gloves have been used, those made of rubber and those made of cotton. If we put rubber gloves on we must, of course, have them fairly tight, and, especially during a long operation, they will clasp the fingers and make them dead, so that nothing can be felt through them. Not only is there the dulness produced by this thick rubber, but you have the fingers numbed by the continuous pressure. If there is no leakage, there is inside these gloves churned up hot sweat from hands which are constantly perspiring and shedding the epidermis, and this, with the germs in it, gets worked up into a fluid. Now supposing towards the end of the operation you happen to prick the glove, out spurts the fluid into the wound, and this fluid has been found to be very septic indeed. I do not myself think that the patient needs protection through gloves, though it may be wise occasionally to put on a pair of rubber gloves to protect the surgeon from the patient, just as is done at post-mortems. For instance, I have put on gloves in cases of phagedenic ulcers in the mouth from which I might otherwise have soiled my hands. With regard to cotton gloves, it has been proposed that the surgeon shall have a dozen pairs of cotton gloves, such gloves being sterilised in one of these chambers. The idea is that you shall put on a pair of these gloves and begin, and directly the fluid soaks through change to another pair of gloves, for it is no longer possible to swill the hands in a basin of antiseptic solution, and so it becomes necessary to have a dozen pairs of gloves. But I think wearing both india-rubber and cotton gloves is erring in the direction of excess of precaution; I do not see that they are necessary, and nothing can compensate for the loss of touch.

When you look round an operating theatre such as this—one which is much used—you see a number of things intended for various purposes, but when we have to think of preparing a private house for surgical operation it is necessary to adapt ourselves to a more simple provision. A private room can be rendered fit for any surgical operation, if there is time, by clearing the room entirely of furniture, including the hangings and pictures, by scrubbing the floor, and seeing that the windows and chimney and doors are properly fitting and will open and close at will; also that there is no bad drainage about the house, or satisfying yourself that it has recently been put in order. Then the room can be disinfected in the way rooms are disinfected after a contagious disease, such as by the sulphur method; but especially by opening all the windows and allowing fresh air and sunlight. By this means we can make the room good for an operation, especially if we can keep it at a proper temperature and at the same time have ventilation. The proper temperature of an operating theatre is about 68° F., that is to say, for comfort. It is apt to go above 70°. You find a feeling of depression if you stay in an operating theatre for an hour with a temperature at more than 70°. Some surgeons say operations should be performed with the theature at much higher temperatures, such as 75° or even 80°, and this is especially advocated abroad. But they do it on a different system. Abroad it is the usual practice to strip the patient and then have the room super-heated. That is very trying to any one who is not used to such heat, and not only trying to the surgeon and his assistants but also to the patient himself, to have to breathe air at such a temperature. The proper temperature I say is about 68°, or at any rate something under 70°.

But when working at such a temperature the patients have to be wrapped up in all parts except the operation area, and covered with hot blankets and hot-water bottles to the extremities, supposing of course that the operation is not to be performed on those extremities. Some go further and have heated operating tables. Though there is a value in them it is an expensive form of procedure, and it is out of the question for private surgical prac-

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tice. Our tables here carry the patient from and to the bedside up and down the lift without moving him. Supposing the private room is heated to 68°, the patient wrapped up in hot blankets with hot-water bottles to the extremities, but not next to the skin, and the bed prepared with hot blankets and bottles, we may again say that we are ready for the surgical operation. You will notice I said the hot-water bottles should not be placed in contact with the skin. Nurses in their hurry have sometimes put the bottles against the skin of the patient, and the patient gets a severe burn from this mistake, and naturally has a good deal to say about the production of this painful burn, which should never have been. In private houses, however, one has constantly to operate where there is no time for such preparation as I have indicated. Under those circumstances what can one do? The best way then is to clear the middle of the room, but do nothing to the walls; do not go near the pictures and hangings. Do not touch the carpet either, except to sprinkle it with water to keep down the dust. Do your operation in the middle of the room, after having set the fire going well so as to keep the apartment warm.

What do we need for a surgical operation? We shall need at least a dozen towels, and a collection of dishes and basins which have been properly scalded and scrubbed with boiling water. Thirdly, we shall want a store of boiled water, that is to say, water which has been boiled for quite half an hour, which should be stored in jugs which have been previously scalded out in the way I have mentioned, covered with towels. With these appliances handy almost any operation can be carried out without much of the other paraphernalia which you find in an operating theatre.

We will now go back to some other points. And first concerning ligatures. There has been an endless discussion, and it is not settled yet, as to what are the best ligatures to use in surgery. The simplest kind of ligature is one consisting of silk, in its various sizes, such as Chinese twisted silk, which may be employed both for ligature and suture. Here are skeins as received from the manufacturers, and they are boiled if you like first of all in a soda solution, and then wound on glass reels. Here are the various sizes kept in a 1 in 20 carbolic acid solution, and they contain no germs which are likely to affect the patient. The only objection to silk is that, especially in the larger sizes, it is apt to remain unabsorbed in certain places, and a long time afterwards to come away. One gets over that difficulty by using as small sizes as possible; also one recognises that silk is not readily absorbed in certain places, and especially in muscular tissue. On the other hand, in the cavities of the body, such as the peritoneal cavity, where there are endothelial cells, silk is rapidly absorbed.

For instance, you can use the largest size silk for ligaturing an ovarian pedicle, and if there is no suppuration you will see nothing more of the silk. If you use silk for a hernia, or for the abdominal wall, it may disappear; or, on the other hand, it may remain for some time and then work out. But it has this great advantage, that it is truly aseptic. For suturing the abdominal wall in stages we use kangaroo tendon; and also wherever silk does not become absorbed, namely, for operations in connection with muscles and intra-muscular tissues. For ligaturing large arteries, and for buried sutures of all kinds, there is nothing like kangaroo tendon. Those two substances for ligatures and sutures will get you through all kinds of surgical operations. Catgut has always seemed to be a thing which, theoretically, should be employed, but the great difficulty is that it apparently has never yet been properly sterilised. All sorts of methods of sterilisation have been employed in connection with it, and each year fresh ones are brought; yet still there is no solution of the catgut difficulty. It is foul to begin with, for it is first macerated sheep's intestine, and is then twisted up, germs and all, in some foul slaughterhouse. That is the history of most of the commercial catgut, and to sterilise it efficiently seems as yet to be impossible, at all events with absolute certainty. Every now and again one will meet with a skein of catgut which will set up in a wound a most virulent infection. The last catgut I used, some years ago, set up in a hernia wound a severe diphtheric inflammation. In this hospital, about Christmas time, we had two patients most carefully and antiseptically operated upon on the same day with a special form of catgut. Both of those patients had the most severe septic inflammation, and nearly died; their lives were undoubtedly placed in very great danger. I have heard surgeons say "I have now got a perfect catgut," but in six months or a year one finds that they do not use it, and when

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asked the reason they say they had a run of bad cases and they had to leave off its use. My opinion is that the sterilisation of catgut cannot yet be accomplished with any certainty. For buried sutures some use fishing gut, others use horsehair, and there have been proposals to use hemp covered with celloidin; that is a proposal of Mr. Mayo Robson,—linen thread for the purpose is a suggestion of Mr. Barker's,—and of course these materials have the merit that they can be sterilised. But they are absolutely unabsorbed, and the knot remains in the tissues as a foreign body, and under certain circumstances it is easy to conceive of its being a source of trouble. I do not say that the most perfect results cannot be obtained by using these substances, but I have had to take out fishing-gut sutures a year after an operation for hernia, when I found that no portion of them had become absorbed; they had remained absolutely unchanged.

I shall just refer, in conclusion, to difficult kinds of sterilisation, namely, that of instruments which are tubular, such as syringes and catheters. It is a very important matter, because these instruments are constantly being employed. A morphine syringe should *always* be cleaned immediately after use. This should be done by syringing it through with 1 in 20 carbolic acid, and then clean and dry the needle by drawing up through it absolute alcohol. This process should be repeated before the syringe is used again. Catheters are also a great trouble. The better way to clean a catheter is immediately after use to get rid of the oil and mucus by washing it and syringing through it with soap and water. Be careful that nothing *dries* on it; if once the material dries on it it is probably hopeless to expect to get it clean. The simplest way to keep a catheter clean is to put it into 1 in 20 carbolic acid tray for one hour, two hours, or a day, in spite of a tendency towards the destruction of the catheter. The common rubber catheter, or black catheter, may be boiled in a saturated solution of ammonium sulphate. Some of the instrument makers manufacture a harder form of catheter, which can be boiled in a soda solution. Besides that, you can sterilise catheters by wrapping them up in gauze and placing them in the steam apparatus. From the point of view of private practice, you can have some such tray as I show you here and place the catheter in it. If you are using a catheter for, say, a prostatic patient, the best way is to keep it under 1 in 20 carbolic acid; even if the catheter wears out quickly, that will be better than having a complication. Take care, however, to swill off the carbolic acid or perchloride if you use that before you pass the catheter, or the old patient will feel much smarting pain.

TUMOURS OF THE KIDNEY.*

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Lecture II.

Renal Tumours in Infancy and Childhood.

In my last lecture I endeavoured to place before you the chief features of the more important forms of renal tumour. We saw that they might be conveniently grouped according to the period of life when the tumour developed, the seat of origin, and the histological characters.

We found that a large proportion were met with during infancy and childhood. Indeed, malignant disease of the kidney is the commonest form of abdominal growth occurring in children. The question is of such interest to the pathologist, and of so great importance to the clinician that I venture to direct your special attention to it.

The fact that malignant disease of the kidney occurred in children was clearly recognised as far back as 1815, when Rance reported a case in a girl of seventeen months. Since that time a very large number of cases have been placed on record, but until comparatively recent years, only meagre information has been forthcoming respecting their structure and true pathological position. I have dealt elsewhere† with the early investigation of these tumours, and need not now trouble you with historical references.

* Substance of a course of lectures delivered at the Post-graduate College and Polyclinic, London, February 14th, 21st, and 28th, 1902.

† 'Renal Growths; their Pathology, Diagnosis, and Treatment,' 1898.

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But we are still in considerable uncertainty as to the relative frequency of the various forms, and have yet much to learn with regard to their ætiology, minute structure, and the best methods of dealing with them.

Most of the growths originating during intra-uterine development and in the early years of life have generally been considered to be of a sarcomatous nature. It is certain, however, that they vary considerably in their morphological characters, and show differences in the degree of malignancy.

It is much to be desired that every growth should be submitted to thorough pathological examination. If our knowledge of the structure of these tumours is to be rendered anything like complete, it will be necessary that microscopical investigation shall not be limited to one portion only, but that detailed examination be made of all parts. Where these growths are recognised early and removed by operation the whole tumour should be submitted to microscopical analysis. In the case of those presenting so-called adenomatous characters it is very desirable that specimens should be obtained in an early stage of their development, and that serial sections should be prepared. If such could be obtained, many points still in dispute would be rendered clearer.

It is only by the study of a large number of cases in which full clinical details are available and complete pathological analysis has been accomplished that we can obtain data which shall secure means for useful differentiation during life.

Sarcomata may arise, as I have already indicated, during fœtal life. Most frequently, however, they are observed in children under five years of age. I find that over 46 per cent. of the cases occurring in childhood are in subjects under three years of age. The female sex seems slightly more liable than the male to the development of these growths. It is generally said that the left kidney is more frequently involved than the right. In many cases the growth is bilateral.

The tumour often reaches immense proportions before seriously endangering the life of the child. It may weigh as much as a third of the whole patient, and, it is said, has reached as much as 36 lbs. Frequently the growth, after becoming clinically apparent, develops rapidly. Sometimes it would seem as though some trifling injury had aroused the tumour to rapid growth. In some cases, however, the progress is comparatively slow throughout.

The fact that so large a proportion of tumours of the kidney occur in early life would suggest that their appearance was dependent on faulty development. Some hold that they arise from embryonic kidney structures, while others contend that they spring from displaced remains of remnants of the Wolffian body. The histogenesis is still very obscure.

Histological study of these tumours as they occur in children show that the following elements may be recognised :—(1) Round-cells occur in practically all specimens ; it is rare for them to make up the whole growth. The cells vary somewhat in size and shape, but usually present typical sarcomatous characters. (2) Spindle-cells are usually present in most specimens, but probably no growth is made up entirely of such. They merely indicate a slightly higher stage of differentiation. (3) So-called muscle-cells are met with in a very large proportion of the tumours occurring in early life. Usually the cell presents more or less striation. The development of these *rhabdo-myo-sarcomata* is almost, if not entirely, confined to young subjects. They were first systematically studied by Eberth and Cohnheim, and later by Neumann, Ribbert, and Wolfensberger, who give references to the early cases observed. The origin and nature of these striated cells have been much discussed. Cohnheim was the first to suggest that they were dependent on a faulty segmentation of the protovertebral somites whereby embryonic muscle-elements were included with those of the primitive kidney. Others have held that they originate in the capsule and pelvis, where muscle-elements normally occur, but there is little evidence in favour of this suggestion. It has been argued also that these striated cells should not be considered as true muscle-elements at all, but rather looked upon as mere ribbing of the surface of a tissue which is not contractile.

Glandular-like structures occur in a large number of the congenital tumours and mixed growths in childhood ; and hence the use of such terms as "sarcomatous pseudo-adenomata," "embryonal adeno-sarcomata," and the like. It is very probable that a more careful and complete examination would show that such elements are even more frequent than has hitherto been generally recognised.

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The occurrence of these glandular-like elements has given rise to much discussion regarding their origin and true nature, and has, of course, added not a little to the perplexities of nomenclature and classification. I think that in all probability their occurrence may have led to some of the early cases of renal growth in children having been described loosely as "cancer," or even under the more definite term of "carcinoma."

The glandular-like structures to which I am referring are not to be confounded with mere stranded renal elements; they are manifestly part of the new formation. Sometimes they appear as distinct tubules, not unlike the collecting tubules of the kidney. Occasionally the arrangement may be distinctly that of an adenoma. The proliferation of the cells may lead to the filling up of the tube or cavity with epithelial-like cells. Careful examination will show that these cells do not arise from the true epithelial structures of the kidney. Frequently the cells of these anomalous glandular-like structures may be clearly traced in the mass of the embryonic cells of the tumour. And still further, complex and compound forms occasionally occur, in which myxomatous, and angiomatous structures can be detected.

But it is unnecessary to weary you with details of the various combinations which give such endless variety to the appearance of tumours of the kidney. I wish, however, to insist that in the consideration of the malignant tumours of the kidney occurring in young subjects we must shake ourselves free from the fettering influence of nomenclature.

In the strict sense, probably a true renal carcinoma or a true renal sarcoma is but rarely met with in children. These tumours are almost always composed of various heterogeneous elements. They are essentially mixed tumours. As Dr. Powell White * has recently insisted, they are tumours of indifferent cells in an imperfect state of differentiation (blastomata). The primary cells of the tumour are to be regarded as indifferent cells derived from the intermediate cell-mass of the embryo. Such cells as they proliferate undergo differentiation, some forming epithelium, others muscle, and so on. This is a conception which undoubtedly meets the facts of the case, and, I think, will help us much in the further study of this interesting group of renal tumours.

* 'Lancet,' February 15th, 1902.

Renal Tumour originating in Adrenal Inclusions—Hypernephroma.

I now wish to allude briefly to another class of tumours which has given rise to considerable confusion and been the subject of much discussion, and even still is not always easily recognised.

In my former lecture I reminded you that aberrant supra-renal tissue was occasionally met with in close association with the kidney in the form of the so-called "adrenal rests." Such "rests," it is well to add, are not confined to the kidneys, but have been met with in the peri-adrenal fat, in the tissues about the solar plexus, in the mesentery, broad ligaments, and adjacent to the ovaries.

The adrenal inclusions usually met with in connection with the kidney appear as small, localised, more or less circumscribed, and occasionally definitely encapsuled, yellowish nodules. It is very probable that certain of the so-called lipomata have really belonged to this class. These "rests" may even themselves include detached uriniferous tubules.

There can be now but little doubt that such included adrenal tissue occasionally proliferates and gives rise to tumours. To such a growth the convenient term of *hypernephroma* has been applied. What the precise influence may be which rouses these dormant structures to malignant growth it is impossible to say.

Unlike the sarcomata we have just been considering, the tumours originating in adrenal inclusions usually make their appearance in adults, and generally between the ages of thirty-five and sixty. Males are said to be more frequently affected than females. Either kidney may be involved, but generally only one organ is affected in the same patient, thus presenting another important difference from the congenital sarcomata.

The growths originating in adrenal "rests" usually present malignant characters. They tend to invade the blood-channels, and give rise to secondary deposits in the lungs, liver, and not infrequently in the bones. Some tumours apparently of this class have been removed by operation, and there is reason to believe that if interference is undertaken sufficiently early life may be saved.

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Hypernephromas vary much in size and shape. They are usually white or yellowish white in appearance, but frequently show hæmorrhagic and degenerative changes. They may be separated from the kidney by a more or less defined capsule, and sometimes are themselves surrounded by the renal capsule. The kidney is compressed, flattened, or actually invaded.

A hypernephroma is made up of stroma and cells. The general appearance often closely resembles that of the cortical portion of the adrenal. The stroma consists of fine, fibrous-like tissue and a network of capillaries. The tumour-cells, which vary much in size and shape are sometimes seen to be placed directly on the endothelium. They may contain glycogen. There seems reason to believe that some at least of the so-called endotheliomas (peritheliomas) have belonged to this class of hypernephromas.

The arrangement of the stroma and cells often gives rise to a somewhat tubular appearance, and hence probably some of the anomalous adenomatous growths which have been described may also belong to this group.

Riesman * has well summarised the principal arguments advanced in proof of the adrenal origin of these tumours:—(1) The situation of the tumour just beneath the kidney capsule, this being the usual seat of displaced supra-renal rests. (2) The character of the cells, which resembles that of the cells of the adrenal cortex. (3) The presence of fat droplets and glycogen in the protoplasm. Although glycogen is not a normal constituent of the supra-renal gland, it is always found in tumours of this organ. (4) The metachromatic character of the nucleolus—that is, its property of staining differently from the nucleus,—a condition never seen in renal epithelium or true renal adenomas, but in adrenal rests and in the cells of the adrenal cortex. (5) The similarity of malignant adrenal and malignant renal tumours of so-called adrenal origin. (6) The absence of any transition between the tumour and the renal tissue. (7) The presence of giant cells, as in simple hyperplastic growths of the supra-renal glands. (8) The existence of an abundant capillary network, such as is seen in the cortex of the adrenal. (9) The tendency of the tumours to penetrate early into the veins. (10) The presence of lecithin in amounts closely approximating those characteristic of adrenal tissue.

It is very difficult to give these growths their precise place in our list of tumour formations. Morphologically, they may resemble either the sarcomas or carcinomas. The histogenetic grouping, of course, depends upon the view we take as to the embryological position of the adrenal. If it is epithelial in origin then we should be warranted in placing these growths among the carcinomas. Until we have more reliable data the term hypernephroma may perhaps be best employed; it is certainly convenient and does not commit us unduly.

In my next lecture I hope to deal with the tumours affecting the normally placed adrenals: and I shall also endeavour to show you in what way certain of the pathological points we have considered may be of service in clinical work.

* 'A Text-book of Pathology,' edited by Ludvig Hektoen and David Riesman, vol. ii, 1901, p. 986.

Supra-renal Extract in the Treatment of Gastro-intestinal Hæmorrhage in the Newly Born.—L. Emmett Holt ('Arch. of Ped.,' April, 1902) reports the case of an infant which began to vomit blood sixteen hours from birth, repeating the vomiting at intervals of a few minutes for about eight hours, when the writer first saw the case. The amount of blood vomited each time was from a few drops to a teaspoonful. Most of it was of a dark brown coffee-colour, mixed with mucus. The general condition of the baby was excellent. There seemed every reason for believing that the hæmorrhage came from the stomach, and it was decided to try the effect of supra-renal extract. One grain of the saccharated extract was ordered every hour, suspended in water. This was given regularly for six or eight hours, after which the dose was reduced to half the quantity, and the intervals were made longer. Twelve hours after beginning the supra-renal the hæmorrhage entirely ceased, and did not recur. The child then began to take its food normally, and when forty-eight hours old was nursing as well as any child. By the fifth day the minute hæmorrhages which had been seen beneath the skin had disappeared, as also those on the mucous membrane of the mouth. In all the child took twelve grains of the supra-renal, and of this ten grains were retained. The entire period of the hæmorrhages lasted about thirty hours. The subsequent history of the child was uneventful.—*Amer. Jour. Obstetrics*, July, 1902.

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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AN ADDRESS

ON

SOME SURGICAL CONDITIONS OF THE KNEE-JOINT.

Delivered before the Wimbledon Medical Society.

By G. R. TURNER, F.R.C.S.,

Surgeon to St. George's Hospital.

MR. PRESIDENT AND GENTLEMEN,—Your secretary suggested to me that I should speak to-night about certain diseases of the knee-joint, and I understood from him it would be desirable that I should refer chiefly to those in which there might be some little difficulty in diagnosis. He was led to make that suggestion to me by the fact that he and I had had under our care a young woman who had a condition of knee-joint that closely simulated tubercle, and yet which subsequently proved to be a sarcomatous tumour of the internal condyle of the femur. It is therefore chiefly on that subject that I shall address you to-night. Of course the time at my disposal will not permit me to speak of all the diseases to which the knee-joint is liable, and I shall therefore direct my attention mainly to sarcomatous tumours, tubercle, and that which imitates tubercle so often, namely, syphilis, and also to certain chronic inflammatory affections of the knee-joint due to other causes, which may be mistaken for one of these conditions.

First of all, with reference to sarcomata, I may say that the case I am going to bring before you in detail to-night was only one of two which I had under my care almost at the same time. One was an instance of sarcoma of the head of the fibula, which, after consultation, I made an attempt to enucleate, but found the external popliteal nerve so involved in the growth that, although I could easily have enucleated the tumour, I must have left the parts paralysed, because I should have removed a large portion of nerve; consequently

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it was thought, seeing that the tumour was no longer confined to the head of the fibula, that it would be better to amputate. That case I shall not further refer to to-night.

The case that I specially wish to allude to is that of a young woman æt. 24, married, an ironer by occupation, whose left knee-joint had been painful and swollen for about five years. It had been worse lately, and was the cause of her limping. She had much pain on walking. Dr. Deas tells me she was under his care, and that there was considerable flexion of the leg on the thigh, which he treated by splinting. A little abscess formed a short time before I saw her, which Dr. Deas opened; he informs me that it was a subcutaneous abscess, and had no communication with the bone. As the leg was flexed and in a faulty position it was straightened, and the knee got smaller under Dr. Deas's treatment, which consisted, roughly, of the application of a back splint and Scott's dressing. When she came to St. George's Hospital there was nothing in the nature of a vaginal discharge. She had had a miscarriage three weeks before, and she was markedly anæmic. In these notes which I have the family history is stated to be good, but I now understand there was a distinct history of tubercle in the family. Certainly the patient looked extremely white and anæmic, though not typically tubercular. On examination it was found that the left knee was the seat of an irregular enlargement; there was a scar where the abscess had been opened, and the chief swelling was on the inner side of the joint. The swelling felt bony, and was not soft or doughy. There was a slight increase of heat, but no redness, and the superficial veins were dilated. The notes say that there was some pulpy swelling, but not much. The patella was movable, and there was no increase of fluid in the joint. No movement was possible, but this was partly due to pain. There was great wasting of the muscles of the thigh and leg, but there was no displacement of bone. Dr. Penrose, my colleague who examined her chest, said there was nothing to contra-indicate any operation which might be thought advisable, although the woman had got very marked anæmia. At a consultation which was held before deciding on operation it was resolved to procure a skiagram of the swelling, and then to perform either excision of the joint if it proved to be tubercular, or amputation if it proved, as we thought it would, to be malignant. The skiagram was accordingly taken, and I have brought it down for your inspection. Although it is not a very excellent print, it shows very clearly the abrupt termination to the line of bone on the inner condyle of the femur; it also shows the irregular enlargement on the inner side, and the place where the sarcomatous growth exists. On February 12th I amputated the femur through the middle of the thigh, using equal antero-posterior skin flaps. There was very little hæmorrhage, but what blood there was was of an excessively watery character. I need say nothing about the amputation, which healed by first intention, and the woman was discharged from the hospital some fifteen days afterwards. I saw her again last October, and, as far as I could make out, the stump was perfectly healthy, but she had got a swelling on the frontal bone and another over the sternum, obviously of the same nature as the original tumour of the femur. That ends the description of the case which I thought I would take as a text for the few remarks I shall trouble you with. And perhaps I might at this stage pass round two other skiagrams of sarcomata involving bone. Here is a sarcoma of the lower end of the radius, very markedly expanded by the tumour; and here is one of sarcoma of the humerus, where also, you will note, there has been a fracture. That was a sarcomatous envelope containing blood, and, as you can see, there is a fracture at the seat of malignant deposit. This patient was subjected to amputation, and eventually died of recurrence in the lungs. I brought these skiagrams to show you how well the sarcomata are depicted by means of the X rays. One point I want to mention, and that is with regard to the microscopic examination. I have a specimen, taken from the case we have considered, under the microscope, showing that the tumour, although it was a central sarcoma, was of a mixed sarcomatous type. It was not, as is so often the case in central sarcoma, of the myeloid form. I have here other microscopical specimens of sarcomata, for which I am indebted to Dr. Rolleston.

There are various points of interest about the case which I want to draw your attention to. First of all, with regard to the duration of the disease. The patient gives us a history of the knee having been bad five years. It is obvious it could not

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have been bad from sarcoma during all that time; probably there was some chronic synovitis. The symptoms had been very much worse for the six months prior to her coming to the hospital. If I may say so, I take it that the sarcoma elected to be deposited in her place of least resistance. How often one sees that to be the case in malignant growths. Let us take such a thing as tumour of the breast. It almost invariably happens in such cases that the woman will give you a history of some blow on the breast, or of some mammary abscess during lactation, or the nipple of the breast may always have been ill developed. It constantly happens in cases of scirrhus of the breast that this part of the body is shown to be the place of least resistance. Let us take as another illustration epithelioma of the tongue. It is common in epithelioma of the tongue to find that the rest of the tongue has been the seat of the chronic superficial glossitis of syphilis, or even of syphilitic ulceration. One is very frequently the precedent of the other. The syphilitic disease has rendered the tongue a place of lowered resistance, and then some trifling irritation produces epithelioma. So much is that the case that probably some of you know that Mr. Jonathan Hutchinson advises that in cases of chronic ulcer of the tongue which do not yield to treatment the patient would exercise a wise discretion in having that part cut out, although there may not be any of the microscopical structure of epithelioma present. At any rate, it shows how malignant disease chooses for its manifestation the parts of least resistance.

Another point in the patient we are considering was the very marked anæmia which I have mentioned. She looked terribly pale and ill, and that might have been attributed to her recent miscarriage. But my impression was that it looked more intense than would be produced by that. Probably it was the anæmia which often accompanies malignant disease, especially when it is attacking more than one place. When she was in St. George's Hospital we did not know she had any swelling except that in the thigh. But her subsequent history shows that concurrently with her departure from the hospital swellings began to form on the cranium and sternum, and these, of course, were irremovable. Only the other day I saw the case of a colleague at St. George's, where it was a question whether the growth was sarcoma of the elbow. One of the prominent features in that case was the marked anæmia of the man. It is true that he had been losing blood from the tumour, but the intensity of the anæmia seemed to be more than could be accounted for from that.

With regard to the diagnosis in the case we are specially considering, the important points are first of all that you can see in that skiagram there was an irregular enlargement on the inner side of the knee; it is not a general enlargement such as you meet with in the diffuse infiltration of tubercle in the knee-joint. That irregularity was very suggestive, and it was increasing rapidly during the time the patient was in the hospital. No doubt it increased rapidly when the bony envelope of the tumour gave way, and it burst out into the surrounding soft tissues. If you look at the skiagram you will see that the line of the femur goes down to a certain point and there stops abruptly. No doubt, when the sarcoma got to the tissues outside the bone, there was the rapid enlargement which became apparent in the short time she was in the hospital before the amputation of the thigh.

Another point which is mentioned in these notes, which are valuable because they were made before the amputation was performed and before the skiagram was taken, and not afterwards, was that there was marked dilatation of the veins. Now marked dilatation of the superficial veins means one of two things: either excessive vascularity of the part, or the existence of pressure on deep veins. It may be in the case of these malignant tumours that both such conditions are present. Sometimes this feature is the earliest suggestion of new growth. I remember well, when I was Mr. Pollock's house surgeon many years ago, a boy being admitted for incontinence of urine. That was the ailment that the case was sent in for. It apparently was one of those obstinate cases of incontinence that give so much trouble. He had been circumcised, and all the other acknowledged remedies for children in this condition had been carried out, but nothing did any good. And one day one found over the sacrum at the back marked dilatation of superficial veins. That was a hint to us that there was some pelvic sarcomatous tumour. Such it proved to be, and the boy died of the malignant disease. One of the earliest symptoms in that case was dilatation of the veins in the superficial parts.

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In the present case, when I saw her there was none of that displacement backward of the bones, or rotation outwards, which so often is seen in tubercular disease of the knee. Dr. Deas informs me that at one time there was a tendency that way, but it had been corrected by his previous treatment. There was no fluid in the joint, the patella was not fixed, and there was no jumping at night, due to what many of us are still apt to call ulceration of cartilage, but which in the present day is thought to be more associated with the bursting of osseous deposit of tubercle into the joint. In such tubercular cases there is starting and jumping of the joint at night. This was not present, neither was there any general pulpiness of the synovial membrane. All these facts were against tubercle. But there was present what you do meet with in tubercle, what Sir James Paget drew prominent attention to many years ago in his clinical lectures, a marked wasting of the muscles of the thigh, a wasting much greater than mere disease could account for. Sir James Paget drew attention to the fact that the wasting which accompanies tubercular disease is very much more than can be accounted for from any mere inaction of the muscles. We could not obtain at the hospital any account of a tubercular family history, but Dr. Deas tells us there was a marked history of tubercle. I do not know that that would influence one very much in making a diagnosis, because it is so difficult to get a history of tubercle from some patients; these people will admit "asthma" and "bronchitis" in the family quite readily, but they will strenuously deny that there has been tubercle. If there had been any tubercle at all in this case it would have been in the form of a local osseous deposit in the internal condyle of the femur, and that is the situation which is most commonly the seat of such deposit. For instance, it is more common there than in the tuberosity of the tibia when it is near the knee-joint. Neither were there, in this particular case, quite the symptoms which we regard as typical of abscess in bone or chronic inflammation of bone, with, perhaps, a little central sequestrum, such as we know sometimes occurs in the cancellous ends of long-bones. There was no question of syphilis in the case. The skiagram at once made the diagnosis a certainty; there could be no longer any doubt about it. You will see from a glance at the skiagram that the condition could be nothing else than sarcoma springing from the interior and eating its way out. The skiagram obviated the necessity of exploration, and it showed us, too, that it was a case for amputation rather than for any enucleation of the tumour. I dare say most of you will remember that it was Mr. Clutton who originally drew prominent attention to treatment of myelomatous tumours which occur at the ends of long bones, and also to the fact that it was not necessary to amputate for them, but that enucleation and scraping the tumour out were sufficient. These myelomata, I believe, are considered by pathologists to be clinically innocent. They occur in the interior of the bones, and also in a very peculiar situation for myelomata, namely, about the tendon-sheaths. Only the other day I removed a small tumour from a young lady's index finger, from the terminal phalanx. That proved to be one of these myelomata of the tendon-sheath, with typical giant-cells. Mr. Bellamy, in the 'Journal of Pathology' of last November, has written a very exhaustive account of myelomata in tendon-sheaths, and he is inclined to attribute the giant-cells to the endothelium of the blood-vessels, and proposes to call them endothelial myelomata, which, he maintains, differ from the myelomata in the ends of the bones. I remember, many years ago, seeing a surgeon amputate a finger for one of these myelomata, mistaking it for an enchondroma or cartilaginous tumour of the finger. He amputated the finger before cutting down on the swelling. When this specimen of sarcoma of the femur, which I removed by amputation, was put into spirit it was shown that the whole of the medulla was studded with sarcomatous growths to within half an inch or so of the section of the femur, and I must say I thought it would not be long before there was a local recurrence in the end of the bone. Last October or November there had been no recurrence, and I was a little surprised to find the stump healthy. It continued so until her death just before Christmas, nearly a year after the operation.

Sarcoma of the end of the bone simulates, then, sometimes a tubercular deposit, and sometimes, when it is in the soft tissues, such conditions may readily be mistaken for abscess. Possibly many of us have cut down on a tumour thinking we were going to open an abscess, but found that instead

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of pus there has been the jet of bright blood which comes from the bleeding of a malignant tumour. I remember doing it myself some years ago. It was a tumour which proved to be an alveolar sarcoma of the transverse processes in the lumbar region. That occurred at the Seamen's Hospital when I was surgeon there. I thought I was going to open a lumbar abscess in connection probably with diseased spine. There was no lumbar abscess present, and when I got down to the swelling it proved to be sarcoma, and its microscopical characters showed it to be of the alveolar variety, which is comparatively rare. In this present case there was no pulsation. These tumours are often so vascular that they pulsate, and this feature of pulsation may be the cause of doubt as to whether a malignant tumour is being dealt with or an aneurysm. I remember seeing a case of pulsating tumour of the head of the tibia that, if it had not been in front of the joint where there are not any very large vessels normally, one would have thought it to be an aneurysm. It differed from aneurysm in various ways, but it turned out to be what it was diagnosed as, namely, a very vascular sarcomatous tumour. That was removed by enucleation, leaving a great cavity in the head of the tibia. This case was not a case adapted for that treatment, because before the wound healed there was a recurrence at the bottom, and the patient had to be subjected to amputation afterwards. There was no need, then, for the differential diagnosis between a sarcoma and an aneurysm, which is made by noting the effect of compressing the main artery above and the tumour itself, and by noticing how many beats of the heart are necessary to fill the tumour again after it has been emptied by pressure; also by comparing the pulse below the tumour with that on the opposite side, by inquiring into the nature of the bruit, the existence or not of lateral expansion of the tumour, and noticing whether the pulsation is real or conveyed. There was no need either for diagnosis between new growth and periostitis. Sometimes that is a matter which may engage the attention of the practitioner or the surgeon. He will have to ask himself, "Am I dealing with new growth or periostitis?" Sometimes it is more than a little difficult to say which is which. A new growth has a better defined and more abrupt border than the swelling of periostitis; the swelling of periostitis generally becomes bevelled into the surrounding tissue. There was no need here, either, for diagnosis between new growth and the callus which forms about the point of union of fractures. There was no history of injury of the lower part of the femur. In fractures of the upper third of the femur sometimes it becomes an important question to settle whether it is simply an exaggerated callous deposit or a sarcomatous tumour with which you are dealing, because, especially since acute traumatic malignancy has been written about, we know that occasionally malignant disease does show itself at the seat of fracture. Sometimes it is consequent upon the fracture and sometimes it precedes it. That case of sarcoma of the humerus was one where you can see in the skiagram by the altered direction of the two fragments that there has been a solution of continuity. I remember seeing a case in which a huge mass of callus in the upper end of the femur was mistaken for sarcoma, and it was proposed to cut down on it and see whether it was necessary to amputate at the hip joint. This was not done, however, and the patient eventually came down to the convalescent hospital here at Wimbledon perfectly recovered, and is now walking about with a very good leg indeed. In fractures when there is much displacement of bone, and in fractures of the upper third of the femur this is constantly the case; there may be considerable swelling, which may be mistaken for malignant disease.

Now I want to say a few words on the diagnosis of ordinary pulpy knee, which is a diffuse infiltration of synovial membrane with tubercle. Can this condition be mistaken for anything else? Yes, it can. It has been well said that syphilis imitates all diseases and initiates none; and it was my fate, some years ago, to see a case of acute syphilitic disease of the knee-joint that almost exactly imitated in every point acute tubercular destruction of the knee-joint. It was that of a sailor who was admitted to the Seamen's Hospital at Greenwich when I was surgeon there. He came into the Hospital obviously exceedingly ill; he was extremely emaciated, with a high temperature, rigors, sweating, with his right knee-joint apparently the seat of advanced tubercular disease. There was a jumping pain at night—those starting pains which are supposed to be typical of ulceration of cartilage. His condition was so grave that it was

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thought amputation would have to be performed. The physician was kind enough to examine his lungs, and he said there was breaking down tubercle at the left apex. The man's condition was so bad that I held my hand before doing anything; in fact I did not think he would stand the amputation. Happily for him some days after admission his throat began to slough, and the soft palate was being rapidly destroyed in this way. That of course was a hint which one was obliged to take, and I put him upon iodide of potassium and mercury, and his knee-joint immediately began to get better, so did his general health, and his lungs cleared up. I suppose he must have had a gummatous deposit about his lungs as well as in his knee-joint. The other knee-joint became affected; he got effusion into both ankles and the left elbow in rapid succession. All these joints cleared up, and eventually perfectly recovered, with the exception of the original knee, which had been bad for a long time before he came in, and which had been destroyed by this process of rapid syphilis. He recovered with that joint ankylosed. The other joints entirely recovered under appropriate antisyphilitic treatment. He had given a very doubtful history of syphilis, but no history of any secondary symptoms at all. That was a case where a diffuse infiltration by gummatous deposit was seen. Localised osseous and synovial deposit of tubercle may also be simulated by syphilis. I have at the present moment a patient in the St. George's Hospital who is obviously subject to old congenital syphilis. She has marks of old interstitial keratitis, she has Hutchinson's teeth, and she has the characteristic facies of that disease. She has also got the left hip-joint ankylosed, apparently as a result of old disease. Whether that old disease is syphilitic or tubercular I cannot certainly say. She came into the hospital complaining of very great pain in and about this hip, and that pain yielded at once to iodide of potassium internally and the rubbing of the part with blue ointment.

Another case I remember was one which was under my care some years ago, where a woman, again with the marked facies of congenital syphilis, was the subject of what looked like tubercular disease. She was put upon antisyphilitic treatment, and over and over again for about two years she improved with rest and this antisyphilitic treatment, making one believe that it was a case of congenital syphilis of the knee-joint. Eventually she got no better in spite of the remedies, and I excised the knee-joint. I had what I removed examined microscopically, and it proved to be tubercular after all. That was a case where tubercle occurred, and where for a time we thought the trouble was really due to syphilis.

Does syphilis ever imitate the class of case which we have been considering, that is to say, malignancy? It does, very closely indeed. I well recollect a case of Mr. Pollock's many years ago of a man apparently suffering from malignant disease involving the lower end of the femur. A consultation was held on the case, and it was decided that in all probability amputation would have to be immediately performed. But before carrying that out Mr. Pollock decided to do what I think all of us ought to do when there is the remotest chance of syphilis being present, give iodide of potassium and rub in blue ointment. By those measures this particular tumour disappeared, and the man got perfectly well. I have also in my mind another case, which was on the Cancer Institute at St. George's Hospital for a good year. The woman came in with a mass right at the top of her head, affecting the frontal bone and further back the parietal bones. This mass was supposed to be sarcomatous. As a result of pressure over both leg centres, which you know are situated in this region, she got paraplegia. She lay for a long time in bed, but she did not do as she ought to have done if it had been sarcoma; that is to say, she did not die. After some considerable time—more than six months—she was put upon iodide of potassium and mercury and rapidly recovered, and for all I know she is living and well now. When she left the hospital the paraplegia was gone, and she was able to walk about well. Here again, therefore, was a case of syphilis imitating sarcoma in the situation which is also involved in the case I have brought before you to-night. This woman, as I have said, had a recurrence on the front of the sternum, and that, again, is a very favourite seat for gummatous deposits.

I will mention yet one other case in which syphilis imitated malignant disease. I remember that a porter at Greenwich got a swelling of the upper jaw and nasal bone. It was thought by my colleague there and myself to be malignant disease; but to make doubly sure he was brought up to a

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London hospital for consultation. The surgeons there thought it was malignant disease, and it was advised that the upper jaw should be removed. The man was something of a philosopher, and settled to have the operation done, but insisted on going for a week's holiday before its performance. His colleague told me that he was really intending to go on the drink. It was suggested to him that, amongst other things, he should drink some iodide of potassium with his alcohol. He came back at the end of the week, having followed this advice, and the swelling had practically disappeared. It was really a case of syphilitic disease of the bones of the face, and not malignant tumour at all.

How are you to distinguish between syphilis and malignant disease, seeing, as I think these cases show, that they so closely simulate each other? Is the history of secondaries of much good? I mean the history of ordinary sore throat, spots about the body, periosteal pains, loss of hair, and so on. I have constantly noticed in the worst cases of gummata that there is often an entire absence of a history of secondary symptoms. Patients who mean to be just and honest with you are perfectly unable to give you a history of secondaries, and one of the worst cases of tertiary gummatous deposits that I have met with was in a gentleman who had a gumma of his kidney and about the veins of the intestine which nearly killed him. The only secondary disease which he had at all was a tiny spot of psoriasis on the upper eyelid. When I was surgical registrar at St. George's Hospital and I had to take notes of a case of gumma, I was often struck by the absence of history of secondary symptoms.

Again, there is the anæmia to which I have drawn attention as being present in this case. This is very marked in some cases of malignant disease, but it is also equally marked in old cases of syphilis. So much is this so that it has been said that iron is wanted as much for this reason as iodide of potassium and mercury in late syphilis. The pallor which is induced by syphilis is proverbial. Gummata, as a rule, are more or less asymmetrical, and if you examine the patient carefully all over you will generally find some mark of the "beast" about him somewhere, though sometimes it is very difficult. I think practically the only way sometimes of proving whether you are dealing with syphilitic manifestations or not is to give iodide of potassium and mercury. If there is any syphilis present, then the improvement from that treatment will be very marked and nearly always rapid. I am, of course, speaking of affections such as we are considering, not of late syphilitic changes in the nervous system. Sometimes such a condition as that is absolutely impervious to any syphilitic treatment at all.

Tubercular knee may be simulated by chronic synovitis due to other causes, and I should like to draw attention to that due to adhesions following traumatism. It is common for traumatism of the knee-joint to occur, and as a result of that there is often synovitis. The leg is very properly kept at rest, and so long as there is fluid in the knee-joint there is no danger of adhesions forming. The fluid prevents the contact of the surfaces. It is after that stage that there is the danger of adhesions, and they constantly do form. Adhesions will not form simply from the joint being kept quiet if there is no inflammation. There must be inflammation *plus* rest to ensure adhesions. When the older treatment of fractures obtained, when both joints above and below the fracture were confined for some weeks in splints, such as plaster-of-Paris or silicate, so long as the fracture was away from the joint one used to see only temporary stiffness when the splints were taken off; the joints were stiff, but there were no adhesions. Adhesions and the inflammation which follows and accompanies them, which inflammation is perpetually set up by their stretching, will sometimes very closely simulate tubercle. I should like to relate here a case which I was brought into contact with last spring. It was that of a young officer who came back from South Africa, where he had been wounded by a shot which passed through the thigh. He had whilst playing polo received a blow from the polo stick over the knee-joint. He was not able to rest or lie up, although there was considerable swelling and extravasation of blood; he had to do work in the trenches around Ladysmith and go through a rough campaigning life. He was eventually invalided and treated by splinting and rest, with the result that when he came over to England he could hardly hobble, and his knee-joint was absolutely fixed. The muscles of his thigh had wasted so as to be almost absent. It seemed to me when I saw him that the right thing was to give him a general anæsthetic and

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break down these adhesions, and employ massage, electricity, and passive movements in a gymnasium. For a time this was successful. But after six weeks or two months, when he was walking about, he did too much, and got a renewed swelling of the knee-joint. There was a history of tubercle in the family, though not a marked history. Before I broke down the adhesions once more under an anæsthetic I thought I would like to have the opinion of another surgeon to share the responsibility. I therefore asked a very eminent surgeon to see the case, and he pronounced it to be tubercular, which was just what I was fearing. He saw the patient again three weeks afterwards and again said he regarded it as tuberculous. But after all it was not so; it was simply a joint which had become chronically inflamed as a result of these adhesions being stretched. That patient is now quite well.

A few words now about breaking down adhesions. We used to be told, when Sir James Paget drew attention to cases that bone-setters cure, that it was necessary to break adhesions rather than to stretch them, and I remember well in my student days seeing adhesions in the knee-joint often broken down, but as a rule without very much success. In fact it used to be said that it was not of much use to break down adhesions in the knee-joint. The reason of this was and is that if adhesions are broken down very thoroughly indeed, and movements made in all directions in the joint where there has been such adhesion, the inflammatory reaction which follows is so acute, and there is so much swelling, pain, and tenderness, that the patient has, of necessity, to lie up, and then adhesions re-form, and the last stage is almost as bad as the first. So if there is to be breaking down of adhesions I would advise that there should be more than one sitting, and the passive movement should be followed by electricity and massage, and the hot-air treatment, which is exceedingly useful for patients after the breaking down has been carried out. To attempt too much breaking down at a sitting will very often give rise to acute inflammation of the joint, and the state of the joint will be as bad as ever. Of course the proper working of the muscles in these cases where there have been adhesions is very important, and I am sorry to say that in London there is only one gymnasium which I know of—and I have inquired of several surgeons—where cripples can go to work the muscles of the leg and arm. I think there should be such a gymnasium at every hospital in London. I am sorry to say we have not one at St. George's. Of course something can be done by means of what bicyclists call the home trainer, a sort of domestic treadmill, and Whiteley's exerciser; but one really wants a proper arrangement of weights and pulleys so that the muscles are perpetually kept in exercise. The result is a greater vascularity of the parts, and the patient is soon able to bring about voluntary movement over the muscles until they get well.

I have come to the end of my time, and have practically said nearly all I intended; but I should like to say a very brief word about hysteria when complicated with a little real disease. Unless the practitioner is carefully on the look-out, hysteria may be easily mistaken for a disease of graver moment, especially if there has been any synovitis or trifling inflammation about the knee. Of course we know that in hysteria the pain is diffuse, and that tenderness, although exaggerated, is not present very often when the attention of the patient is directed to something else. It is very difficult to lay down any guiding rules in words in reference to the diagnosis of hysteria, and all of you must be perfectly well aware of this. You use your common sense, and by various methods arrive at a conclusion as to how much of the symptoms are real and how much fanciful. Hysterical joints are not always seen in people whom you would expect to be the subjects of them. They are sometimes met with in children who are very young.

I have met with a hysterical joint in a young highly intelligent girl about eight years old, whose father, himself a mighty Nimrod, had insisted on her riding high-spirited horses; and have seen a hysterical ankle in a child about six.

If by any remote chance you should be taken in, or pretend to be taken in, by an hysterical patient, it may possibly come about that by thus gaining her confidence you may effect a cure where others of superior diagnostic skill have previously lamentably failed.

I have no time to-night to say anything of internal or other derangements of the knee, to touch upon new methods of treatment made possible to us by modern surgery, but there is one old friend—often the refuge of the destitute—I

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must caution you against, rheumatism. I have seen the pain of a popliteal aneurysm all but treated by antirheumatic methods, and alas! how often has the acute diffuse periostitis of children been mistaken for acute rheumatism. Careful examination of the joint and its neighbourhood must precede all diagnosis, all treatment; and even this, as I have endeavoured to show to-night, may not at once throw light on the more difficult and dark places of knee-joint disease.

A DEMONSTRATION OF MEDICAL CASES IN THE WARDS OF CHARING CROSS HOSPITAL.

JUNE 12TH, 1902.

By H. MONTAGUE MURRAY, M.D., F.R.C.P.,
Joint Lecturer on Medicine.

GENTLEMEN,—This patient, æt. 52, is an asylum attendant. He was admitted because of total blindness of the left eye and shortness of breath; or rather, he came complaining of those defects. The history is as follows:—He has been told that at the age of four he had an attack of rheumatic fever. From that time he had no illness or accident until five years ago, when he received a severe blow on the left eye, but without any recognised impairment of vision. He has been in the habit of taking three pints of beer a day, but no spirits, and of smoking two ounces of strong tobacco a week. There is no history of syphilis. For the last year he has suffered from morning cough, anorexia, slight breathlessness, and an occasional sensation of faintness. This brings us to the onset of his present illness, which occurred ten weeks ago, with a sudden attack of dyspnœa while walking. He went to bed, but only for a day or two. The breathlessness soon improved under treatment. About three weeks after this attack, on rising in the morning he found that he was blind in the left eye. Since that time there has been no marked change in his condition, except that the dyspnœa returns on exertion, and his feet are occasionally swollen. You will notice that he is a powerful and well-nourished man, and plethoric in appearance. On admission his tongue was slightly tremulous, his temperature was normal, and the urine contained a trace of albumen. These signs have persisted since his admission. His left eye and his heart furnish the only other indications of disease. The left pupil reacts somewhat less readily than the right; at the present moment it is dilated with homatropine. When you examine the fundus presently, you will find the characteristic signs of embolism of the central artery of the retina; that is to say, the optic disc is pale, its edges are slightly hazy, and the retina is a little opaque. The veins and arteries are alike small; if anything the veins are smaller than the arteries, which is unusual. On examining his heart you will find that the impulse is only just palpable, and that the apex-beat is practically in its normal position. On admission the deep cardiac dulness extended a little too far to the right, but this is now less distinct. At both apex and base a faint systolic murmur could be heard—more distinctly at the base than at the apex. The pulse is small, regular, not delayed, and of a uniform rate of about 88, and the arteries are not degenerated. The lungs and abdomen present no abnormal physical signs.

It has been said that sudden blindness of one eye is sometimes the first indication of heart disease; that disease of the aortic valves may be so slight and cause so little secondary change that the whole condition passes unobserved until the embolism of the retinal artery occurs. But undoubtedly that is unusual, at any rate in the experience of the ordinary physician. Embolism of the central artery of the retina is usually a complication of cerebral embolism, and it is one of the things by which one diagnoses that a patient has embolism and not hæmorrhage. The appearances which you will see in the eye really demand no explanation. The diminished size of the arteries and veins is due to deprivation of their blood-supply, and of course the blindness is quite easy to understand, for whenever such a delicate structure is deprived of its blood-supply its nutrition is so interfered with that its functions are permanently lost. You know that if a nerve-cell is deprived for half an hour of its blood-supply it dies; and the retina is practically as delicate as a nerve-cell. The haziness of the retina which you observe is due to œdema and cellular infiltration. In most cases there is more of the former than the latter. When only a branch of the artery

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has been blocked you still get total blindness at first, but the part which is supplied by the unblocked portion of the artery recovers its function, and permanent blindness only affects that part supplied by the blocked vessel. It sometimes happens that even when the main artery is blocked the circulation may be ultimately restored. In rare cases it may be restored directly from the breaking up of the original clot; more often it may be restored indirectly by anastomosis; but in both cases the restoration of the circulation comes too late to restore the nutrition, and so it is not followed by corresponding restoration of sight. The opacity of the retina disappears in two or three months, and the optic disc gradually undergoes atrophy. So if you see a case in which the vessels are very small and the disc atrophied, and there is a history of sudden blindness, absence of opacity of the retina does not militate against the diagnosis of embolism. In this case the embolus is almost certainly derived from the heart; there is no evidence of aneurysm of any of the great vessels, and a clot carried from the aortic valves most commonly passes into the left carotid artery. At the same time it must be remembered that there was no evidence of active endocarditis at the time the blindness occurred. There is no reason to suppose that the blow on the eye in any way predisposed to the condition.

The condition of the heart in this man is of much interest. From what I have said about the character of the embolus and the position of the apex-beat you will gather there is practically no hypertrophy. This condition in a man who has aortic disease and embolism resulting from it suggests three possible explanations. The first is, that the condition is quite recent, and that there has not been sufficient time for hypertrophy to occur. The second is, that the alterations in the valves are insufficient to cause marked hypertrophy, that is to say, there is a noise due to the presence of vegetations, but there is no real obstruction, and therefore no hypertrophy, the hypertrophy being the measure of the obstruction. The third explanation is, that the impaired nutrition of the heart prevents compensatory hypertrophy, in which case failure should come on as an early result. This man has had some definite evidence of cardiac failure for the last year—there has been breathlessness, a sensation of faintness, and so on,—and therefore I think we are probably right in accepting the third explanation of the absence of cardiac hypertrophy. He probably has some degeneration of his myocardium. The cause of this is uncertain. It may be due to alcohol or some agent affecting metabolism, or to disease of the coronary arteries impeding the entrance of the blood into or circulation through them, since disease of the coronary vessels is frequently associated with similar changes in the aortic valves.

If the arteries are diseased at all at their orifices or along their course, and free circulation of the blood through them is prevented, compensating hypertrophy of the heart cannot take place, and failure will soon result. It may be asked, and, indeed, was asked, whether pulmonary embolism would not have accounted for the attack of breathlessness ten weeks ago. But there is no discoverable cause for pulmonary embolism, such as venous thrombosis; and embolism will not account for the signs of cardiac failure which he has had for the last year, whereas cardiac failure does appear an efficient explanation of the whole symptoms. His trouble cannot have existed very long, because, as is usual in such cases, he was thoroughly examined before he was appointed asylum attendant; and he says that he has been in the habit of taking active exercise, such as cricket and the like, for twenty-four years.

The treatment he is having at present is directed to improving the nutrition of his arteries; he is having the iodides of potassium and iron, and his cardiac condition is being mainly treated by rest. At the onset of embolism of the retina, the treatment is much the same as in embolism of the brain; it is, if anything, more important that he should be kept at rest, for if a small clot has been carried into the eye a larger clot may be detached and plug one of the important arteries in the brain.

This man æt. 73 came to the hospital for dysphagia which had been progressing for two months. The cause of death of his parents is in neither case known, but one sister died after two years' illness from a growth obstructing the œsophagus. Our patient had rheumatism twenty years ago. There is no history of syphilis or of the swallowing of corrosive poison. The first thing the patient noticed was the regurgitation of a portion of his

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food immediately after it was swallowed. Until a month ago he could swallow fluid with ease, but even the attempt to do this is now accompanied by slight regurgitation. Until a fortnight ago he was able to swallow a slight amount of solid food, but only after complete mastication and in very small amounts. Thus you see a month ago he began to have difficulty in swallowing fluids, and a fortnight later he could not swallow solids at all. Therefore during the last fortnight his diet has been limited to fluids. At the present time a portion of everything he takes regurgitates, and you will see here the matter which is regurgitated. It is alkaline, and consists of food mixed with mucus and saliva. He has never had any pain or spasm, nor has the seat of obstruction appeared to him to vary. He has never brought up any blood, but has lost much flesh, and is now losing it at the rate of about one pound per week. Small, hard, shotty glands can be felt above both the right and left clavicles. The tongue is dry and brown. The passage of a bougie is arrested ten and a half inches from the teeth, which locates the upper end of the obstruction five and a half inches above the cardiac orifice of the stomach. No bougie has been passed through the stricture. The right vocal cord moves rather less readily than the left. The heart's action is weak, and the arteries are much degenerated. There is some bronchial catarrh. The right rectus abdominis muscle seems to be more rigid than the left, but no enlargement of the liver can be made out. There is no evidence of any intra-thoracic aneurysm, such as pulsation, irregularity of pupils, or other sign of pressure. In this case, then, there can be but little doubt that the patient is suffering from a squamous epithelioma of the œsophagus. With regard to the other possibilities, cicatricial obstruction is very unlikely. It is a much rarer disease, and there is no evidence of anything which would cause simple fibrous stricture, for we can find no history that he has swallowed any corrosive fluids or that he has had syphilis. Besides, the onset is much more rapid in this case than is usual in those due to cicatricial obstruction. It takes some years for this to develop, and the whole progress of the case is much slower. It is sometimes said that in malignant disease a temporary relief of the obstruction may occur, owing to the degeneration and detachment of superficial portions of the growth. This is undoubtedly true, but it is perhaps more correct to say that the occurrence of such temporary relief is not evidence against malignant disease, for certainly in the majority of cases even temporary amelioration is not met with. The relief which is sometimes noticed is more often due to the careful and systematic feeding adopted when the disease is recognised than to actual reduction in the size of the growth. After a short interval the progressive wasting is again marked.

In this patient we have not only his age and the rapid progress of the obstruction to help us in the diagnosis, but we have also the presence of affected glands,—a comparatively rare, but very valuable sign. In the majority of such cases the glands are not felt to be enlarged. This patient does not suffer any pain, and never has done so. That is an important thing to remember. Pain is much more marked in spasmodic stricture of the œsophagus than it is in malignant stricture, or, indeed, in any form of organic stricture; and when it is present in organic stricture is often due to accompanying spasm. The implication of outlying parts in this case is comparatively slight. The right recurrent laryngeal nerve seems to be the only one affected. As regards treatment, this is at present a question of feeding. It is impossible to get any tube through the obstruction, and therefore he is merely being fed upon arrowroot and milk, some of which passes the obstruction. It is not proposed to adopt any other measures so long as he can get sufficient nourishment in this way, but should the obstruction increase surgical interference may be advisable. In many cases of growth in the œsophagus, perforation of the canal, with resulting cellulitis or extension to the pleura, pericardium, or trachea, occurs before the obstruction becomes complete. Of course, in a case of this kind, it is useless to perform gastrostomy unless it is absolutely necessary to enable feeding to be carried on. It is often better even to keep a patient alive by rectal feeding until the complications I have referred to render it unnecessary.

There are one or two things which are worth alluding to while considering a case such as this. It is a good general rule never to pass a bougie until the existence of an aneurysm has been practically excluded, because if an aneurysm press on the œsophagus you may rupture the sac, how-

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ever carefully you may pass the bougie. The second point is that in the early stages of malignant disease there is sometimes so much pain and spasm as to give rise to the impression that the case is only one of spasmodic stricture. For instance, cold food may be found much more difficult to swallow than warm food, which is one of the features characteristic of functional spasm of the œsophagus; or there may be attacks of cramp-like pain, such as are seen in spasmodic œsophageal stricture. The best way to remove the doubt is to pass a bougie; and there is no bougie which gives you as much information as an olive-headed one. The old-fashioned gum-elastic bougie of the same diameter all the way down was of less assistance in determining the position and extent of the stricture. In any case of stricture pass the bougie down and note the distance from the teeth at which it becomes arrested; then withdraw it if it will not pass further. Then use, with extreme care, a smaller bougie, until, if possible, you find one that will just pass the stricture. On withdrawing it note the distance from the teeth when the olive-headed end is first gripped by the stricture. This will tell you the position of the lower end of the stricture. In spasmodic stricture, the point at which the bougie is arrested varies, often even during the examination, whereas in organic obstruction the point of arrest never varies, though this point tends to progress very slowly in accordance with the extension of the disease. A certain amount of irregular spasmodic contraction may occur in organic stricture; but when this is so there will still be a fixed stricture in one place. There are other symptoms which are of less importance from the point of view of diagnosis. Of these the most important is that marked loss of flesh is rare in functional cases, and that when it occurs the symptoms have generally persisted for many months, or even for years.

Here is a boy æt. 11 years. His history is that he has had dyspnœa for the last three months. There is no history of any acute illness except measles. He presents the typical appearances of cardiac dyspnœa, but the cyanosis, rapid breathing, and obvious distress are very much less than they were when he was admitted rather more than a week ago. There is œdema of the lungs, but no ascites, and very little œdema of the legs. The heart presents the typical signs of mitral regurgitation—a dilated heart and insufficient systole. The impulse is diffuse, and the apex-beat is difficult to localise, but is best felt in the sixth interspace, one and a half inches outside the nipple line, showing the enormous size of the heart. There is the usual typical systolic murmur conducted into the axilla, the usual accentuation of the pulmonary second sound, and, in addition, a systolic murmur distinctly audible in the pulmonary area. The pulse is frequent but regular. The veins on the surface of the thorax are distended, and the flow of blood is from below upwards. There is no intermission of pulse or fall of pressure during inspiration, and no clubbing of the fingers. The liver is very much enlarged; its surface is smooth, its edges are even, and both are tender on palpation.

There are three things in this case which are of interest. The first is, that in children it is not uncommon in cases of cardiac failure to find that the enlargement of the liver is a much more prominent change than œdema of the lungs or general dropsy. I cannot offer you any explanation. Some say it points to mitral stenosis being present as well as regurgitation. There is no evidence of mitral stenosis here. Others say that the enlargement of the liver is proportionate to the actual amount of tricuspid regurgitation. We have no certain method of testing this. Certainly a systolic murmur is very distinctly heard over the sternum, and that may be due to tricuspid regurgitation. I think it is really more accurate to say that œdema of the lungs and general dropsy are less easily produced in children by cardiac failure than it is to say that the enlargement of the liver is more readily induced. In children the lungs seem to possess a resisting power which they have not in the adult. I do not think the ultimate prognosis is influenced, but the immediate prognosis is better when, although the liver is large, there is no œdema of the lungs.

The present condition of the mitral valve furnishes a sufficient cause for the enlargement of the heart, and the cardiac failure equally obviously depends upon the inability of the heart to keep pace with the growth of the patient and with the continued results of the valvular lesions. The valve goes on becoming harder and stiffer and less pliable, and therefore the need of hypertrophy becomes greater. There is a limit to the hyper-

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trophy of any organ, and that limit is very soon reached when the progressive growth of the child and the progressive disease alike demand a continually enlarging heart.

There is in this boy no history of any previous disease. This absence of any clear history of previous endocarditis is really of little importance. In many of the worst cases of heart disease in children there is no history of previous disease. The reason is probably twofold. In some cases it is forgotten, and in some it has passed unrecognised. I think perhaps one reason why so many of the serious cases of heart disease in children have no history of acute illness is that the malady passed unrecognised, and that there was no prolonged rest or treatment at the time of the original trouble.

These cases are analogous to those of Bright's disease in which the children are said never to have had scarlet fever, but their parents may perfectly remember when "the skin came off in lumps." Rheumatic attacks may be equally neglected or only remembered as "growing pains." Those are the cases which are not taken to the doctor for treatment at all in the early stages. It is therefore a good general rule to examine the heart in all indefinite febrile attacks occurring in children.

There is another point of interest about this boy. I mentioned when speaking about his case that there was no intermittence in the pulse, or fall in the arterial pressure during inspiration. Although both these phenomena may occur in perfect health, constituting the so-called "paradoxical pulse," they constitute the most characteristic signs of so-called chronic indurative mediastino-pericarditis, and this is one of the causes of cardiac failure with enlarged liver in a child. The onset of the disease is frequently insidious without any symptoms of acute illness. Sometimes it arises from the glands in the mediastinum or from other mediastinal structures, and affects the pericardium secondarily. In other cases it commences with pericarditis, and affects the mediastinum secondarily. But in whatever way it arises it leads to strangulation of the great vessels, and especially of the veins, by cicatricial tissue, and impairment of the heart's action by pericardial adhesions. The change in the pulse already described is not attended by similar changes in the cardiac sounds, but there is usually swelling of the veins on forced inspiration. In this patient a deep breath makes no difference to the distension of his veins. Again, in cases of mediastino-pericarditis systolic retraction of the chest may occur, and even deficient local expansion during inspiration. The enlargement of the liver in these cases is not wholly due to passive congestion, but partly in some cases at least to a cirrhotic condition as well, which has given rise to the suggestion that it is due to syphilis. Some perihepatitis is occasionally found to account for the tenderness and the ascites. It is a question whether this boy has any such condition. In the majority of cases of mediastino-pericarditis there is enlarged heart and liver, but no valvular disease. But if there is marked distension of veins during inspiration the diagnosis is fairly certain, even though valvular disease be present. When, however, there is marked valvular disease, it seems to be going out of the way to assume that there is any such rare condition present, unless the positive evidence is strong. In ordinary mitral regurgitation you do not generally find so much venous distension as this boy has had; but enlargement of the liver may be present. The prognosis is a little different in the two classes of cases. If the heart is not very large, the patients may do fairly well for some time; but if the child has a large heart, and has come to the end of his capacity for hypertrophy, any further attack is necessarily disastrous. Indurative cases may occasionally go on for some years. I have seen two boys with livers down to their iliac fossæ, and they had attacks of cardiac failure. They were kept in bed, got well, and ran about again. But although the course is longer the final result is practically the same. This patient is taking the ordinary cardiac tonics and stimulating expectorants for the œdema of his lungs. Children with this condition rarely reach adult age. Cases of cardiac failure occurring in adults never give any history of cardiac failure during childhood unless the disease is congenital.

We have had in this hospital Covent Garden porters who have had a clear history of rheumatism and endocarditis, but who have, nevertheless, done their work for twenty years before cardiac failure developed, and you know that their work is very arduous. But that, of course, meant that they had no very serious valvular defect at the outset, and that, therefore, there was not much compensatory hypertrophy needed; they have never got anywhere near the limit between compensation and failure. They were, moreover, probably both temperate and free from syphilis.

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TUMOURS OF THE KIDNEY.*

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Lecture III.

Adrenal Tumours.

In my last lecture you will remember I referred to those renal tumours which were believed to have their origin in misplaced supra-renal tissue.

To-day I wish to deal in the first place very briefly with the primary adrenal tumours, for although their consideration does not strictly come within the bounds of my subject, yet it must be admitted that when of sufficient size to give rise to a perceptible tumour, they cannot usually be distinguished clinically from the true growths of the kidney.

Benign Tumours of the Adrenals.—The suprarenal bodies may undergo diffuse hyperplasia, and this glandular proliferation may be sufficient to produce distinct general enlargement. For such a condition the term "adrenal goitre" has sometimes been used, but it is in several ways objectionable, and should be discarded. Virchow termed the condition *struma suprarenalis.* But not infrequently the adrenal hyperplasia is more or less localised, or at all events assumes the form and shape of what we are accustomed to consider a tumour. To such the term of adenoma may be applied.

It will therefore be seen that the so-called adenomatous formations of the adrenals occupy a position which it is not always easy to determine. In some instances the so-called adenoma is little more than a circumscribed hyperplasia, while in others the morphological characters and general arrangement of the cells and stroma would, according to some, merit the application of the term carcinoma.

It has been customary to recognise two chief forms of adrenal adenoma:

1. Multiple, where numerous small, yellow, nodular bodies occur, usually situated in the cortex, often ill-defined, and generally with no distinct capsule; they are made up of cells which closely resemble those of the normal adrenal cortex; marked fatty changes are frequently apparent.

2. Single adenomas are occasionally met with in the post-mortem room. I show you here on the screen a good example. It gave rise to no symptoms during life. These growths do not always involve the whole organ; they may, however, reach considerable dimensions. They often present an abundant stroma infiltrated sometimes with round-cells. There may be a rich supply of vessels, and even an angiomatous condition may be met with. The cells are frequently arranged in a more or less columnar form, and in many instances closely resemble the cells in the zona fasciculata and zona reticularis of the normal adrenal. Sometimes spaces are apparent, lined, or even filled with small cubical cells, and even distinct papillomatous in-growths may be met with. The cells often show pigmentary changes, and undergo fatty or other degeneration. Hæmorrhage into the substance of the growth may occur. The chief microscopical features are indicated in the specimens I am able to show you on the screen and in the drawings and microscopic preparations which I have placed for your inspection. The so-called adenoma is the usual form of simple adrenal tumour met with, but fibroma, lipoma, glioma, angioma, and lymphangioma have been recorded.

Malignant Growths of the Adrenals.—The number of thoroughly investigated cases of primary malignant disease of the adrenals is very small, and hence our present knowledge is sadly lacking in precision and completeness. A progressive atypical proliferation of adrenal tissue does, however, occasionally occur, but under what precise circumstances we cannot say. Certainly these malignant forms are met with both in young subjects and adults. The older cases were usually recorded under the head of "cancer," but with our present views concerning the development of the adrenal, and considering also the morphological characters and pathological tendencies of the growths, it is now customary to class them with the sarcomata. These growths may reach very considerable dimensions, encroach on the adjacent kidney, infiltrate neighbouring parts, extend along veins, and give rise to secondary deposits and marked cachexia. The cells are often very irregular in size and shape.

* Substance of a course of lectures delivered at the Post-graduate College and Polyclinic, London, February 14th, 21st, and 28th, 1902.

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Sometimes large multinucleated giant-cells are met with. Frequently they are made up largely of round-cells, which do not differ materially from those met with in sarcomata elsewhere.

Unfortunately at the present time we have not sufficient data to state clearly whether any special clinical features occur in connection with adrenal growths. As already indicated, on physical examination the tumour is usually indistinguishable from that of one originating in the kidney.

Much speculation has arisen regarding the possibility of growths composed of adrenal tissue forming a secretion in any way similar to that produced by the normal organ. It is interesting to note that in some of the cases of adrenal sarcoma distinct nutritional changes have been observed during life. Thus instances have been met with in children where abundant growth of the hair has occurred, and even cretinoid characters have been observed.

Perirenal Tumours.

It is not my intention at the present time to deal with the tumours which arise in the immediate neighbourhood of the kidney, but I must remind you that perirenal growths occasionally occur, and it may be impossible to distinguish them clinically without exploratory incision. Indeed, sometimes in the case of a retro-peritoneal sarcoma the growth may invade or surround the kidney, so that removal can only be accomplished by the loss of the kidney also.

In these lectures dealing with the pathology of renal tumours, it has not been my purpose to direct special attention to clinical features, but rather to indicate the bearings of pathological studies on the diagnosis, prognosis, and, to some extent, the treatment of the various morbid conditions we have considered.

It will be well, therefore, to refer very briefly to certain pathological points which are of some particular assistance in forming a correct clinical opinion.

One of the most important evidences of renal growth is enlargement or "tumour." It is often the first indication in children. It always occupies the lumbar region, except in those very exceptional instances where a movable kidney becomes the seat of growth. When on the right side it may be obscured by a large overlapping liver. Sometimes it becomes adherent to the liver, and is then readily mistaken for a growth of that organ. The reniform outline is frequently retained even when the growth has reached considerable dimensions. When, however, the capsule has been penetrated, and nodules give an irregular surface, care must be taken when the tumour is on the left side that in a cursory examination any such irregularity is not mistaken for the notch in an enlarged spleen. It is often said that the colon is invariably found in front of a renal tumour. It is well to remember that while this is often the case, yet occasionally, and especially when the growth is on the right side, the intestines may be displaced to the inner side of the tumour, and, indeed, may be adherent to it. Usually a distinct renal tumour presents but little mobility, and often there are extensive adhesions. Many of the rapidly growing sarcomata, and some of the malignant adenomata, are very soft and vascular. In these sometimes a sense of fluctuation may be obtained, and occasionally distinct pulsation, and even a soft blowing systolic murmur has been heard over such growths.

I have already referred to the important bearing of the point of origin of the tumour on the progress and symptomatology of the case.

The passage of blood in the urine, in the absence of evidence of disease in the lower urinary tract, is often a very important sign of renal tumour. Indeed, it is probably met with at some period in something like 50 per cent. of the cases. It is often said that hæmaturia is less frequent in children than adults. This is easily understood when you remember how, in not a few of the specimens I have shown you, the pelvis was not markedly invaded until the growth had reached a considerable size. In some cases hæmaturia is an early sign. Such was the case in this preparation of malignant papilloma of the renal pelvis. The quantity, of course, varies greatly according to the pathological condition. The benign growths rarely cause hæmaturia. Some have held that it is more persistent in carcinoma than in sarcoma, but if such is really so it is probably because the pelvis is more likely to be invaded early before the renal secretion has been altogether arrested or the ureter completely blocked. It is well to remember that the blood may clot in the pelvis of the kidney and ureter, preventing the appearance of blood for a

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time, and later appear as shaped clots, sometimes vermicular in form.

Pain in these cases is most variable, and is a symptom of but little value, but I would remind you that the passage of clots or portions of growth along the ureter may give rise to attacks of renal colic. Occasionally nerves in the neighbourhood of the kidney may be involved. It is remarkable, however, to what immense size these tumours do develop in children without causing more than slight discomfort.

Some French observers have considered the occurrence of a secondary or symptomatic varicocele an indication of some service. It certainly may occur as a consequence of pressure upon and obstruction to the spermatic vein by the tumour.

In connection with our modern views respecting the pathology of uræmia, it is interesting to note that toxic symptoms are not usually conspicuous in these cases.

From what I have said regarding the size, weight, situation, and extent of these tumours, you will readily see that pressure symptoms may be of much importance. In the case of children I have seen practically the whole of the abdominal cavity filled by the growth.

The urine in renal tumours may, as I have already said, contain fragments of the growth, and thus afford valuable aid in forming a pathological diagnosis. Where deposits of melanotic sarcoma occur in the kidney the urine may contain melanin.

Renal tumours extend (1) by local extension, or (2) by general infection. In the latter case involvement occurs by means of the lymphatic channels, or by venous invasion. The latter is the more frequent. In one case I found a sarcoma extending along the renal vein into the inferior vena cava and projecting into the right auricle. The growth not infrequently projects into the renal pelvis, and may grow downwards along the ureter.

Secondary deposits occur most frequently in the abdominal glands, lungs, and liver, but sometimes have most extensive distribution.

As I have frequently pointed out in the course of these lectures, renal tumours of almost all kinds are liable to undergo various regressive changes. Fatty and myxomatous degeneration is common. Hæmorrhage into as well as from the growth is frequent. Patches of necrosis are sometimes met with.

The rate of progress and duration vary greatly, and depend in great measure, as I have attempted to show, on the essential nature of the tumour.

The means whereby a complete diagnosis can be arrived at, I think you will agree, must come in great measure from pathological data.

It is not for me to dwell at the present time on the methods of differential diagnosis. Much of this is well known to you, and the study of the specimens I have placed before you will to some extent have indicated those pathological features which will assist in the task of discrimination.

But in arriving at a complete diagnosis, I may remind you it is necessary to distinguish:

1. Renal tumours from non-renal enlargements.
2. Renal tumours from other forms of renal enlargement.
3. One form of renal tumour from another.

Renal tumours may easily be confused with adrenal growths and perirenal formations. They have also to be differentiated from omental and mesenteric growths, peritoneal tumours, and even some forms of localised peritonitis. Such affections as cystic disease of the kidney, renal tuberculosis, hydatid disease, hydronephrosis and pyonephrosis, renal abscess, nephrolithiasis, and nephroptosis have been mistaken for new growths of the kidney. And among the conditions which have been confused with renal tumour are growths of the liver and gall-bladder, splenic enlargements, ovarian and uterine tumours, fæcal accumulations, pregnancy, ascites, and phantom tumours.

I do not intend entering upon the question of treatment. The records of nephrectomy, it must be admitted, are hitherto disappointing.

It is to be hoped that as our knowledge of the various forms of renal tumour increases, we may obtain more definite information regarding the precise characters of these growths, and thus be able to differentiate them more fully clinically, and perhaps render more effectual aid than is possible at present.

Large Placenta.—Perret ('Bull. de la Soc. d'Obst. de Paris,' No. 8) describes a placenta 64 centimetres in circumference, 6 in thickness, and weighing 1,850 grammes. Examination showed that it was hypertrophied and macerated, but no vascular lesions suggestive of syphilis were found. —*Amer. Journ. Obstetrics*, July, 1902.

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ULCERS OF THE TONGUE.

A Clinical Lecture delivered at St. George's Hospital, June 10th, 1902.

By A. MARMADUKE SHEILD, M.B., F.R.C.S.

THERE was a patient in Fitzwilliam Ward about a month ago who had half of the tongue removed for epithelioma. Some of you may remember the case. At all events I thought the notes would prove a useful text for the consideration of a very important subject—the different sores which occur on the tongue, and their differential diagnosis and principles of treatment. When speaking of sores on the tongue I would include the whole of the organ, the sides and the floor of the mouth; in fact, the neighbouring parts in immediate contact and relation with the tongue itself. There are a large number of ulcers which may affect the tongue: some of them are of secondary importance, I mean in so far as regards their power to destroy life; others are carcinomatous, one of the most serious and dreaded diseases which can afflict humanity.

Now I suppose that if I were to consider and classify ulcers of the tongue in order of frequency, the first variety I should bring before your notice would be the lesions of syphilis affecting that organ. Syphilis affects the tongue very variously, far more so perhaps than books would lead you to expect. The first variety of syphilis of the tongue is the primary sore, the so-called Hunterian chancre. It is rarely ever seen in this country, and is of minor importance. I can only recollect to have seen one case. But when primary syphilis does affect the tongue it is marked by the usual characteristics of a rapidly forming sore of peculiar appearance, with enormous swelling and indurated glands under the jaw, with a copious dusky rash upon the skin. You will probably never see a case of the kind.

The other varieties of syphilis affecting the tongue are found in all stages of the disease, and, indeed, a bad syphilitic tongue may last a patient

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his lifetime—sometimes better, sometimes worse, never quite sound and well. Syphilis is often found to affect the tongue about three to six months after infection, when the rash is fading upon the body, and when infectious "mucous patches" are so frequently found around the anal orifice. The great characteristic of secondary syphilis affecting the tongue is the formation of white patches of epithelium, with small superficial ulcers, always made worse by rough and jagged teeth. The friction of the tongue against rough teeth leaves painful excoriations on the organ, and these eventually give rise to ulcers. Early syphilitic ulcers are almost invariably multiple, and are found especially on the side of the tongue. They are commonly associated with ulcers on the side of the mouth, the tonsils, fauces, or soft palate. You will observe in all these ulcers the same characteristic—the epithelium is thickened and white in patches. In fact, I might say that you never get syphilis affecting the tongue without the epithelium being thickened and diseased; it is one of the great characteristics of the malady.

In the later stages of syphilis comes another variety of lingual disease, the so-called gummatous disease of the tongue, common, and often very difficult to diagnose from cancer. As regards gummatous disease of the tongue, it may occur very many years after the primary infection. The latter may be completely forgotten, and the patient often is a man of undoubted respectability, whose social position may seem to defy your diagnosis. This form of disease of the tongue usually commences as a hardish, painful lump in the substance of the organ—I am speaking of a typical case,—and afterwards disintegrates, forming a deep ulcer, the base of which is "foul" and sloughing. But the edges, the base, and surrounding parts lack the excessive hardness which is so characteristic of cancer. Here, again, the epithelium in the neighbourhood of the ulceration is seldom or ever healthy; you almost invariably find whitish patches and deep fissures, and these signs point out quite clearly the true origin of the malady. I have never seen a case of syphilis of the tongue with healthy surrounding surface. I look upon the thickened white epithelium with cracks and fissures as one of the most important diagnostic features of syphilis of the tongue. The presence of scars of healed ulcers on the tongue or throat is significant, and the parts should be narrowly scanned under a good light. You will often read that gummatous disease of the tongue first attacks the centre of the organ. This is true, but the situations of ulcers of the tongue are absolutely no certain guide as to their true nature. A deep syphilitic ulcer may occur in any part of the organ.

In all these various affections the question of history is often one of great importance. But when the ulcers occur, as such affections do, in persons of the upper class and in all positions of life, you must be guided by what you observe on the tongue more than by anything the patient will tell you, because a man of undoubted probity of life may have suffered from syphilis early in life, and may absolutely have forgotten that he ever had anything of the kind the matter with him. Be guided, therefore, far more by what you see than by what you hear.

These are some of the main diagnostic characteristics of syphilis of the tongue. I must just mention that very often cases occur which are extremely difficult to diagnose, especially cases of gummatous ulceration, even though you try the microscopical test or apply the treatment for syphilis and await results. As regards the microscopical test, I advise you at once to disregard all statements made as to the appearance of "scrapings" of these ulcers. It used to be the custom to scrape the base of the ulcer, and some people pretended that they could recognise so-called cancer-cells. But this test is quite unreliable. Perhaps a better mode is to take out a small portion of the edge of the sore under cocaine, and examine it under the microscope; but even then the appearances are often exceedingly obscure and vague. The changes which occur in epithelium in chronic inflammation at the borders of old ulcers are closely akin to epithelioma, and difficult, if not impossible, to differentiate. Therefore, the microscopical test does not give you the help that you would expect. As regards the treatment test by iodides, it is of some value, but I think should seldom be persevered in for more than three weeks. A truly syphilitic case will show manifest improvement in that time. So much, then, for the syphilitic ulcers of the tongue.

And finally remember that syphilis is so frequent a cause of ulcers of the tongue that you

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must ever think of it, even in the most unlikely individuals, when such cases are presented to your notice. Syphilis is a strange disease, and curious stories concerning it occur in human lives. A man or woman in any station of life may have syphilis, and it may affect the tongue or it may not. I speak strongly with regard to this clinical fact, but not too strongly.

I will next speak of that variety of ulcers of the tongue generally described in books by the name of dyspeptic, or aphthous, or herpetic. Perhaps the most appropriate name is "dyspeptic." As regards these ulcers, even now the true pathology of them is hardly understood. In people who are out of health, very much run down, overworked, and suffering from chronic indigestion, it frequently happens that small, multiple, painful ulcers form on the tongue, resembling very much herpetic ulcers on the skin. They are excessively tender, far more so than syphilitic ulcers. I have seen several cases which I believe carries out the idea that these ulcers are really due to contagion. You know it is very common for people to get small ulcers on the lips. A little herpetic rash forms on the lips, and it afterwards scabs. I have seen several cases which lead me to believe that these so-called "dyspeptic" ulcers of the tongue are really produced by contagion from one person to another in the act of kissing. The great diagnostic sign of these ulcers are their multiplicity, their rapid formation, and extreme painfulness. They are very small, seldom larger than a No. 1 shot.

I pass on to a form of lingual ulcer which is of surpassing importance, because it is so often overlooked in practice, I mean the traumatic ulcer of the tongue. Nothing is more frequent than for an ulcer of the tongue to form from injury, and that injury is almost invariably produced by a "jagged" tooth when part of it has been broken off, and the tongue is perpetually abraded by the rough edge. A badly fitting tooth-plate is as serious. This injury may develop into an ulcer so like cancer that it is very difficult to diagnose between the two conditions. In elderly people such ulcers, if neglected, almost invariably become cancers. Rarer forms of traumatic ulcers of the tongue are those produced by scalds from hot fluid, or wounds from pieces of fish-bone and the like, which have injured the tongue. The burning and friction caused by the stem of a clay pipe may act in the same injurious manner. A characteristic of these ulcers is their position at the side of the tongue, and the detection of the exciting cause. The extraction of a rough tooth may cause the rapid healing of a very dangerous-looking ulcer. Never irritate such sores by rubbing them with caustics. This treatment only precipitates the danger of cancer. It is the worst thing you can do.

To complete this subject I will very briefly speak to you of two cases which came under my own personal observation within the last two years. One was that of the wife of a gamekeeper down in the country, quite a young woman, under thirty years of age, who had at the back of the tongue a sore which had been produced by constant friction of the tongue against a rotten, jagged, molar tooth. She showed the sore to a medical man in the neighbourhood, and he did what is, alas! a very common thing to do in such cases—the jagged tooth was left, and the sore rubbed with caustics. This went on for some six months or so, and in the meantime a rapidly growing cancer formed in the site of the ulcer. I went to see this unhappy woman, and found the root of her tongue infiltrated with cancer. I could do nothing in the operative way, and she had to die a miserable death. Large doses of morphia were administered in order to render her unconscious to pain. This is only an example of many sad cases which occur as the result of an absolute want of knowledge in not extracting a rough tooth which rubs upon the tongue. As a contrast to this case, in Princes Ward about two months ago there was an old woman who came in with a hard, indurated mass at the side of the jaw and tongue which looked exactly like epithelioma. We noticed that she had a jagged tooth rubbing the parts, and we decided to extract this before doing an operation, which would have necessitated the removal of a portion of the jaw. The tooth was taken out, and within a month the whole ulcer had healed and the induration disappeared. The mass was inflammatory, not cancerous. Such a case shows strikingly the beneficent action which sometimes influences these ulcers by the extraction of an offending tooth. I trust you will forever remember these cases, and never fall into the dangerous errors I have thus plainly indicated.

I am putting off the all-important subject of

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cancer to the last, and I will next shortly allude to some rare ulcerations of the tongue and its neighbourhood.

1. You know of the disease called pemphigus, where large blebs form over the body. You may get in this disease the same kind of action occurring in the mouth and under the tongue, and on the conjunctiva. The blebs under the tongue may be associated with painful ulcers. When the eruption on the body is large and associated with enormous crust formation and intractable ulceration, and the same action occurs on the tongue or in the air-passages, the prognosis is most serious.

2. In association with this subject, old feeble persons sometimes get very painful sloughing ulcers under the tongue. One cannot explain their occurrence, and they are most difficult to cure.

3. Next comes tuberculous ulcer of the tongue. I do not recollect having seen more than two or three instances of this malady. The ulcer is very characteristic in appearance, just like a tuberculous ulcer elsewhere, with a ragged margin and a yellow caseous discharge; such ulcers are generally found in people in the advanced stages of tuberculosis. You may find these ulcers in an emaciated patient, with cavities in the lungs, loss of voice, and, perhaps, tuberculous larynx.

I must take up your time for a brief space to discuss the hæmorrhagic ulcer of the tongue. This is an exceedingly rare disease, and a very fatal one. It receives little notice from authors. I myself have seen three instances, in each of which the disease ran a similar course. The ulcers occurred under the tongue, and spread slowly despite all remedies. Blood constantly oozed, notwithstanding the use of all sorts of astringents. The ulcer was covered with flabby granulations of a deep livid-blue colour, from which blood constantly flowed away. This disease is associated with some bad and acute form of sepsis. All these three patients died in the same way—they got hæmorrhagic eruptions all over the skin, with high fever, and died in a condition of acute sepsis. The integument became sallow and jaundiced, and post-mortem putrefaction set in with such rapidity that the whole body crackled with gas within a few hours of death. These cases are excessively rare, but you will remember in your future practice that if ever you get a patient with a hæmorrhagic ulcer on the tongue, and it begins to bleed, and the bleeding continues despite all remedies, the case is of the utmost gravity and danger. The sooner you have the best aid the patient can afford, the better for all parties concerned.

The last case of this curious malady which has come under my notice was that of an apparently healthy man about forty-five years of age. He had the best aid London surgery could afford. Nothing checked the bleeding. This patient died in about a week with hæmorrhages all over the skin, and putrefaction was so extreme that within a few hours the whole body was emphysematous. Was this due to the presence of the *Bacillus septicus?* A bacteriological examination was never made in this case, and the possibility of anthrax occurred to my mind afterwards.

I now pass on to the affections of the tongue which I have put in a special class by themselves, because they are perhaps the most important of all, namely, the cancerous ulcers of the tongue. One of the first things a patient thinks of when he or she gets an ulcer of the tongue is the possibility of cancer. A most dreadful thought! Frequently the patient will put off showing his tongue to a medical man as long as possible in fear of the verdict being confirmed. Now recollect this point about this serious disease. Cancer seldom engrafts itself on a healthy tongue. If there was no excessive smoking, no syphilis, no rough neglected teeth, no ill-fitting tooth-plates, probably there would be little cancer of the tongue. For these reasons, lingual epithelioma is far more common in men than women. Cancer of the tongue being produced by irritation, the exciting causes are principally as follows:—(1) Irritation of the tongue by smoking, especially hot clay pipes which irritate and burn the organ. (2) Irritation of the tongue by neglected, ill-fitting tooth-plates or neglected, jagged, carious teeth. (3) Irritation of the tongue produced by obstinate chronic syphilitic changes.

Cancer of the tongue is usually a disease of late life. There are exceptions to all rules. Sometimes cases will occur in quite young men of thirty to thirty-five years old. Instances have been seen in this hospital in men of under thirty, and the disease runs such a virulent course at that age that it is often not worth while interfering surgically.

Diagnosis of cancer of the tongue may be easy enough or may be exceedingly difficult. In a typical case the ulcer occurs generally towards the

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side of the tongue, the edges of the ulcer are hard, indurated, and "gristly," as can be well perceived by gently pressing the pulpy portion of the index finger upon them. The disease is associated with severe shooting pains in the nerves which supply the tongue; reflex pains in the distribution of the fifth nerve, about the jaw, and in the auditory canal are common.

The tongue is prone early to become fixed in the floor of the mouth, the cancerous infiltration extending into the muscles. Next, the glands under the jaw and in the neck are very early enlarged and infiltrated. The extreme hardness of the edges of the ulcer is very characteristic, and the base may be covered with granulations, and slough.

There is another variety of epithelioma that I must draw your attention to, and it is of great importance, because of its greater amenity to treatment. I mean papillomatous or "warty" epithelioma of the tongue. This variety is well illustrated by the man in Fitzwilliam Ward, who had at the side of his tongue a heaped-up growth about as large as half a walnut. These cancers are similar to a papillomatous epithelioma that grows on a scar or an old burn on the leg. I have seen precisely similar growths on the glans penis. In the case of the tongue they are commonly engrafted on old, obstinate, syphilitic disease, the epithelium being thick, white, and fissured (leukoplakia). A large number of papillomatous epitheliomata of the tongue, when they are removed, never grow again. The prognosis is much more favourable in this form of the disease than in that other which is associated with rapid and deep infiltration of the organ and the infection of the glands down the neck.

I have spoken regarding the value of the microscopical test in cancer, and caution you not to rely upon the nature of the infiltration-cells as proof positive of that disease. I have also told you that the iodide of potash and mercury test should not be persisted in too long. You will find in practice that occasionally cases will arise, especially in syphilis of the tongue, where an ulcer forms, and where it is next to impossible to be sure whether it is a syphilitic ulcer or epithelioma occurring on a syphilitic basis. In old ulcers of this nature it is far better to remove the ulcer before it becomes cancerous than afterwards. I cannot impress this clinical truth upon you with too great force. If you are called to a case of obstinate ulceration of the tongue with thickened epithelium, and it resists all remedies, advise that that portion of the tongue be removed before the too probable cancerous infiltration has occurred. It is only, of course, a few sensible patients who will submit to this. As a general rule the ulcer heals under remedies, but breaks down from time to time. At length cancer arises, and then it may be too late. It is an aphorism in surgery that if an irritating sore on the tongue cannot be got rid of by any other treatment, it is far better to remove this potential cancer before further and more serious mischief has occurred. So much, then, for the diagnosis of cancerous ulcerations of the tongue.

Now we will go on to say a few words about the treatment of these affections. First, there are general rules regarding the treatment of all ulcerations of the tongue which must be very carefully observed. For want of observation of them, I have known treatment repeatedly fail. Rule No. 1 is this:—Pay attention to the teeth. I have already spoken about the ill effect of jagged teeth, and how, when these teeth exist, their edges should be filed or they should be extracted. More than this, it is important that the teeth be daily cleansed with an aseptic mouth wash. Rule No. 2:—All smoking must be absolutely prohibited. Time after time I have had cases of ulceration of the tongue brought to me by medical men, who say, "I have tried this and that remedy, and somehow or other the thing always breaks out again." I say to the patient, "How many cigars do you smoke a day?" "Not many now; I used to smoke six or eight, but the doctor only allows me two at present." That is quite sufficient to arrest the healing of any ulcerated tongue. Not even a cigarette should be allowed. Rule No. 3:—There are certain articles of diet which must be carefully avoided in ulceration of the tongue—mustard, pepper, curry, spirits, especially mustard and pepper; but indeed any article of diet which irritates the tongue must be discontinued. Rule No. 4:—In cases where ulceration of the tongue is excessively painful, it is always my practice to give the patient a slate and make him write his wishes, and forbid him to talk in case he may rub the ulcer against his teeth and irritate the part.

How are you to treat syphilitic ulcerations of the tongue—which I have said are so common?

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The general rules for treating syphilitic ulcers are these:—The patient should be placed under the influence of iodide of potassium, and in gradually increasing doses. The prescriptions and "vehicles" are so numerous that I cannot attempt to describe them here. A mercurial application should be applied to the tongue. One of the best for common purposes is, equal parts of the mercury and chalk powder with tragacanth, which is applied as a paste to the part and slowly swallowed off. Calomel fumigation to the affected part is also most useful. There is just one caution here. I do not think it is wise to apply mercurial fumigation if iodide of potassium be taken at the same time. Sometimes an iodide of mercury may form locally, which is rather an irritant.

Another excellent local application in these cases is the cyanide of mercury. Of course it is a very active poison, and is to be carefully used. My own method of applying it is to make the lotion of ten grains to the ounce with glycerine and water. I apply it myself thoroughly with a brush to the affected parts of the tongue, making the patient keep it on as long as possible, and afterwards rinsing out the mouth with water. I have found in some cases of ulceration of the tongue that sometimes iodides fail to do good, and if so mercury often proves very beneficial, especially if the mercurial is applied by unction, or injected immediately into the muscles of the buttock, a course which I am very fond of adopting. These are only some of the various methods which you can employ in syphilitic ulcerations of the tongue. In cases where cracks and fissures occur with much local thickening of epithelium, chromic acid or salicylic acid may be used. Chromic acid can be applied, in the proportion of 20 per cent., with a small mop or brush, and salicylic acid in the like proportion. These applications are painful, and must not be too often repeated. In cases where the ulcers are very tender or painful you may combine with any application to the tongue a certain proportion of cocaine, which is often beneficial, especially before the swallowing of food. Such, then, is the treatment that I advise you to adopt in cases of syphilitic ulceration of the tongue. The earlier in the course of the disease the tongue lesions occur, the greater the need of placing the patient on a mercurial course of a prolonged and careful nature.

Next I pass on to the treatment of so-called dyspeptic or aphthous ulceration of the tongue. These cases are often extremely difficult to treat. First, you should bear in mind the general rules already promulgated for treating an ulcer of the tongue. Next, you will find the greatest benefit accrue in these cases in opening the patient's bowels with mild mercurials, by giving the ordinary remedies for dyspepsia, more especially bismuth, and by sedative applications locally. Do not rub the ulcer with caustics. Weak solutions of cocaine may be applied to the mouth before food. Often these troublesome cases are most rapidly improved by attending to the general health. A change of air to the seaside or the country will often cause ulcers to heal which you have been vainly treating in London for many weeks. Chlorate of potash in full doses is also very useful. Finally, remember you must exclude syphilis very carefully before treating a case of ulcer of the tongue as merely "dyspeptic."

As to the painful ulcerations of the tongue which occur in old people, they are very remarkable, and especially on account of their obstinacy to treatment. I do not understand their true pathology, and I know only two remedies which cure them, and those are quinine and opium. In the Medical Society's 'Transactions' you will find a case related by myself, which occurred in Charing Cross Hospital, where an old man was practically dying with this form of ulcer of the tongue. Every other remedy had been tried without avail. The sores had even been cauterised with fuming nitric acid. Large doses of opium were given, and completely cured the disease. You begin in these cases with either solid opium or laudanum—twenty drops of laudanum three times a day, then add five drops each time after the first day until the pupils thoroughly contract under the opium. As the doses are increased you will find these painful ulcers of the mouth, which have resisted all other treatment, heal as if by magic. In pemphigus with lingual ulcerations you will sometimes find arsenic of great benefit. In one of these cases I have known quinine and opium of benefit.

With regard to tuberculous ulcer, that, of course, needs sedative treatment also. I have treated one such case by the curette and lactic acid, after cocanization, with marked benefit.

I have spoken of hæmorrhagic ulcers of the

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tongue, and noticed their extreme fatality. I think, if ever such a case were brought under my notice again, I should destroy the ulcer most thoroughly with the actual cautery at a black heat. I believe this treatment would give the patient the best chance of life. A bacteriological examination for anthrax should at once be instituted. These cases are so rare, and I know so little about them, that I can advise you only imperfectly. Among the local astringents are turpentine, adrenalin chloride, and "iron wool;" but all these may prove quite unavailing.

With regard to treatment of cancer of the tongue, we all know that the only remedy is to be found in surgery, and in early surgery. Here comes in the immense importance of the subject to any of you who enter general practice. When a patient in the early stage of carcinoma shows an ulcer or soreness of the tongue or floor of the mouth to the general practitioner, if the latter is a judicious man he will save a great calamity in the patient's family by at once seeking or adopting surgical measures. If he is a foolish man he will say, "It is nothing," or will rub the sore with "lunar caustic." The patient will be allowed to go on smoking, and thus his destruction is ensured. The successful treatment of these cases depends on their early treatment, and the only treatment is by surgery.

It is often difficult in cases of cancer of the tongue to know whether they ought to be operated upon or not. The greatest differences of opinion exist on these matters. The extent of an operation for cancer of the tongue is often in proportion to the age and sanguine temperament of the operator. The contra-indications for operations, I will tell you briefly, are as follows:—If you get a case where the tongue cannot be protruded from the mouth, it is a bad case; the muscles and floor of the mouth are infiltrated, perhaps to the epiglottis. If the infiltration has extended so far back in the tongue that you cannot get your finger behind it on to soft and healthy tissue, it is a very bad case; it means that if you amputate the diseased part of the tongue you leave cancer elements behind which will soon reproduce the disease. If the disease has extensively invaded the glands of the neck so that you get infiltrated masses fixed to the jaw and pharynx, the outlook is very bad. If the disease is of the acute variety, with rapid ulceration and infiltration of the tissues of the mouth, especially towards the floor of the mouth, and if this condition occurs in a man under forty years of age, the patient will seldom live long, whatever you do by operation.

It comes to this, therefore—the favourable cases of cancer of the tongue for operation are the hard papillomatous epitheliomata occurring on the side of the tongue, which are slow and chronic in formation where the organ is mobile and can be protruded from the mouth; and the older the patient, the more withered the patient, the more shrunken the patient, the more successful, probably, will be the result of the case.

Indeed, cancer of the tongue follows the rule of growth of cancers all over the body. Cancers in old people are more chronic than in young, vigorous people. I have seldom seen much good result from treatment of cancer of the tongue in persons under forty years of age; it is generally a very virulent disease, and rapidly kills them.

Supposing you have to operate upon cancer of the tongue, recollect that the operation must be of a very free nature. One of the worst things you can do in such an operation is just to "snip out" a cancerous ulcer of the tongue. The affected part of the organ must be very freely removed. I advise you not to attempt such an operation unless you can command the services of a first-class anæsthetist who is well acquainted with the steps of the operation that you are going to do. I always operate upon these cases in the lateral position, the side of the affected part of the tongue being uppermost. All the blood runs into the dependent cheek, and thence to the floor, and there are no embarrassments to respiration, which so often occur when the patient is placed in other positions.

Another method of doing these operations, and a very good one, is by performing laryngotomy and stopping the upper part of the larynx with a soft sponge. The anæsthetic is given through the tube, and the latter is removed so soon as sensibility returns. In cases where the growth extends far back, and the posture is dorsal, I think highly of this method.

At the operation you cannot be too careful to securely ligature the main vessels as they are cut. Snip the muscular fibres very slowly, and seize the lingual artery where it is spurting and tie it on the face of the stump with fine, strong silk. I think this is better than tying the lingual artery in the

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neck. The best operators cannot always find the lingual artery in the neck, and so the patient may get bad hæmorrhage when the tongue is divided. Also vessels of considerable size sometimes enter the tongue from other arteries, so the old rule of tying vessels where they are cut is the best to adopt. I am aware that here I run counter to the practice of many operators. After operation, of course you want to have the patient's mouth constantly washed out with some antiseptic lotion or spray, talking must be prohibited, and he must be fed carefully with a feeder. The one great danger in these cases is septic pneumonia, which is always avoided by cleansing the mouth.

I do not like the plan advocated in these cases of dissecting up flaps of mucous membrane and sewing them over the cut surface of the tongue. It is a method which certainly enables the tongue to heal far more quickly, but it is accompanied with the risk of leaving infected tissue behind.

With regard to removal of the tongue with such instruments as the *écraseur*, I have very little to say. The use of the *écraseur* is an especially risky proceeding, because it crushes the surface of the tongue, and I know from the experience of past days it by no means always certainly stops the hæmorrhage. I am never happy in leaving a tongue case, unless I have with my own hands tied the lingual with security on the face of the stump.

When glands are infected in the neck, the case is far more serious. If the mass is fixed, with extensive infiltration, and especially if suppuration is associated, little or no good will ensue from operating. In early gland contamination very free operating must be practised. Large triangular flaps, containing the skin fascia and platysma, must be turned down, and the triangles of the neck dissected free of glands and all surrounding tissues in conjunction with them. In late cases this is impossible, and though you may relieve the glands apparently you leave behind infected tissue, and return is certain. Operations for cancerous glands in the neck should be "secondary" to the tongue, and as a rule not done at the same time.

In cases where the tongue cannot be removed, you can do a great deal to comfort the patient by keeping the mouth aseptic with carbolised washes, painting the sloughing ulcer with carbolic acid, and applying the powder of orthoform with morphia.

Now as to the question of dividing the lingual nerve in cancer of the tongue. That is a most useful proceeding, and I have done it over and over again in severe cases of cancer of the tongue with the greatest benefit to the patients. In this operation the patient is anæsthetised, the sloughing part of the tongue is cleaned, and the incision is made below the last molar tooth of the lower jaw. With a small hook you then hook up the lingual nerve and cut a piece out of it. This will give great relief in the pain experienced, especially in the anterior surface and sides of the tongue.

Finally, in most cases you must have recourse to morphia. Morphia is a bad drug to commence. It undermines the moral power of the patient, and often seems worse than the disease. At the same time the avoidance of severe pain is one of the things one must aim at, and this can only be effected by large doses of morphia.

Those of you who have attended this lecture, before you leave this room, remember my parting word—do not neglect ulcers of the tongue in their early stages. If you have reason to believe that they will develop into cancer, then have them removed while there is yet time. Cancer in its early stages, especially in old people, and when engrafted upon syphilis, is a hopeful disease if only it is taken early and freely operated upon.

The Prognosis and Treatment of Chronic Nephritis.—The condition of the skin has a direct bearing on the prognosis, so also has a severe degree of anæmia; or the frequent recurrence of uremic attacks is a bad prognostic sign. Rochester's plan of treatment has as its basis the *improvement in elimination and nutrition*. As regards elimination, we should not try to stimulate into activity an organ that is inflamed or degenerated by the use of drugs that excite the functional activity of that organ. Selection of the diet is of great importance, and he gives *milk* a prominent place. *Systematic exercise* must be obtained by massage when active exercise is not advisable. He treats the anæmia by *oxygen-gas inhalations* and the administration of *iron*. The bowels are to be kept open by salines or calomel. He has devised a method of giving a *steam or hot-air bath at home*—*Philadelphia Med. Journ.*, August 30th, 1902.

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WITH DR. E. W. AINLEY WALKER AT GUY'S HOSPITAL.

GENTLEMEN,—We commence to-day the first series of the Gordon Lectures.* It would not be appropriate to pass over the occasion without some reference to that fact, and to the enlightened generosity which has provided the members of Guy's Hospital with fresh facilities for carrying on the investigation of disease and of the means by which disease may be prevented or be arrested when it has developed.

It is to the benefaction of a governor of this hospital that we owe the foundation of these lectures which bear his name, and the endowment of a research laboratory for the advancement of medical science and the ultimate advantage of humanity at large.

In the present course of lectures we shall begin by considering the local reaction which we call inflammation. We shall in our earlier meetings examine the phenomena which it presents, with especial regard to their relation to local infection. From this we shall proceed to discuss the general reaction to infection in general; and shall finally consider the facts and meaning of one of the commonest and most intimate accompaniments of both these reactions, namely, the process known as fever.

THE GENERAL PATHOLOGY OF INFLAMMATION.

The term inflammation is applied clinically to local conditions, which are usually said to be characterised by certain definite clinical signs. Those signs have been handed down from the time of Galen, as calor, rubor, dolor, tumor, and, as Celsius very properly added, *et functio læsa*—heat, redness, pain, swelling, and impairment of function.

Of these five so-called cardinal signs, pain, and in many cases also, impairment of function, is not a sign at all but a symptom, and is not objectively determinable excepting by its effects; and *none* of them is at all essential to the process, since any one or more may be entirely absent in cases of undoubted inflammation.

Thus, as regards pain after section of a nerve, for instance, the sciatic of a dog, an inflammation of the foot can be produced at least as readily as before, although no pain accompanies it. The continuity of sensory nerve-tracts is therefore not essential to inflammation; and, as you are aware, unless special measures are taken to protect the eyeball, a most acute pan-ophthalmitis is liable to follow removal of the Gasserian ganglion of the fifth nerve for trigeminal neuralgia, although and, indeed, *because* the sensory impulses normally produced by irritating foreign matter entering the conjunctival sac have been entirely cut off.

Both heat and redness, being dependent on increase of blood-supply, are absent from the inflammations of non-vascular areas, as, for example, in experimental inflammation of the cornea, or of the tail fins of certain embryos.

Swelling is necessarily absent in the early stages of acute inflammation of non-expansile tissue, such as bone. And both pain and impaired function may be so completely absent, in certain syphilitic inflammations, for example, that the subject is completely unaware of any morbid change. On the other hand, heat, redness, and swelling may all be present in a marked degree without the existence of any inflammation, as in the physiological activity of glandular and erectile structures.

It follows, therefore, that though inflammation is usually accompanied in the higher animals by these classical signs, which are both useful and necessary for its clinical description, they do not in any way assist us to a definition of the thing itself.

It is therefore necessary in pathology to get rid of the idea of inflammation as a condition marked by certain signs, and we must recognise it as a process, or succession of conditions or changes which are usually, but by no means invariably, accompanied by the signs in question. In this way we shall include in the process of inflammation all those events which, under suitable conditions, may result in the four classical signs, pain, heat, redness, and swelling, but must seek a definition founded on ætiology, that is, causation, rather than on symptomatology.

Such a definition was given by Burdon Sanderson in his well-known article in Holmes' 'System of Surgery.' He says it is the succession of changes which occurs in a living tissue when it is injured, provided that the injury is not of such a degree as to at once destroy its structure and vitality; or, as

* Gordon Lectures on Experimental Pathology.

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he himself briefly expresses it, the process of inflammation is the response to injury.

Professor Adami has comparatively recently endeavoured to set aside this definition as being too narrow, and has substituted for it a definition of inflammation as the local manifestation of the attempt at repair of actual or referred injury to a part, or briefly the local attempt at repair of actual or referred injury. Here the last clause is introduced in order to include certain at present insufficiently explained experiments on the production of inflammation by *hypnotic* suggestion. This definition has the objection, which Adami himself foresaw, that its expression is highly teleological, that is to say, that it attributes purposive action to the cells concerned which are said to "attempt" repair. A teleological expression is always a misfortune, and is only permissible, if at all, in general description, where it may save a good deal of circumlocution; but in a definition it is highly inappropriate. Moreover, the definition includes too much, for repair can be both "attempted" and completed without the occurrence of any inflammatory reaction whatever. Indeed, even the definition of inflammation as the response to injury is itself in this respect somewhat too wide, since it makes no distinction of the kind of injury concerned. For it has been shown conclusively that purely mechanical injury does not give rise to inflammation. This was proved by Kocher, of Berne, who found that almost any mechanical injury may be done to bone, for example, if done asceptically, without producing inflammation; an observation upon which depends the justification of osteoclasty as a surgical procedure.

Where inflammation follows in such cases, it is due invariably to septic infection. This was proved as follows by Chauveau:—In the south of Europe it is customary, instead of castrating cattle, to simply twist the testicle, rupturing the spermatic cord. Fatty degeneration of the testicle ensues, but inflammation never follows this purely mechanical injury. But Chauveau found that if active septic material were previously introduced into the circulation, the procedure always led to local suppuration.

Mechanical injury, therefore, by itself does not give rise to inflammation. But a purely mechanical injury is in general so unusual an event, that we may accept the definition of Professor Sanderson—by far the best that has been given—without further question, merely remembering the existence of the exception I have stated.

John Hunter (died 1793) was the first observer who investigated the phenomena of inflammation, which he regarded as being brought about by vascular changes. He taught that inflammation primarily consisted in an increase in size of the blood-vessels and a formation of new vessels in the part concerned. Indeed, he drew a sort of parallel between this process and the increase in size of the external carotid arteries, which he found to occur in stags at the period when their horns are growing rapidly. And he supported his position by the statement, which was certainly accurate, that the heat of inflamed parts never exceeds that of the circulating blood. In his book on the blood, inflammation, and gun-shot wounds, published in 1794, the year following his death, Hunter says, "The act of inflammation would appear to be an increased action of the vessels;" and again, "The part inflamed becomes to appearance more vascular than in the natural state, and it is probable that it is really so both from new vessels being set up in the inflamed part. . . . Besides, the vessels of the part are enlarged."

The view of Hunter remained in general acceptation for the next fifty years. But with the discovery of the microscope and its application to histology by Malpighi came the recognition of the importance of the tissue elements in physiological and pathological processes. This was first emphasised by Sir William Bowman, then by Goodsir, of Edinburgh, who recognised the changes taking place in tissue-cells in inflammation, and finally by Virchow, who established in his 'Cellular Pathology' the doctrine which is now universally recognised, that all the events of physiology and of pathology alike are primarily dependent on the normal or abnormal activities of the body-cells. Virchow, in fact, taught that the whole process of inflammation consisted in a succession of changes in the tissue-cells of the part concerned.

Finally, we come to Cohnheim, who introduced the true experimental method of observation in pathology. This consists in attempting to determine the relation between a number of concomitant or subsequent events by varying the conditions one at a time and noting exactly the effect of such variations upon the result obtained. This is the

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only accurate method of investigation. In disease, on the other hand, the conditions vary from the normal, several and sometimes many together, and in different directions, and the result is therefore complicated, so that we are unable to determine with certainty which of the variations present is the cause of any given effect. A good illustration of this is found in the increase of urea which, as we shall see, occurs in fever. Here the variations from the normal are pyrexia and disorder of nutrition, and we cannot determine from an examination of disease alone whether the pyrexia or the disorder of nutrition, or both, are to be held responsible for that increase. But in experiment as introduced by Cohnheim, it is possible by varying only one condition at a time to see exactly its relation to the gross result.

Cohnheim applied this method to the investigation of many of the problems of pathology with remarkable success. And any one who cares to see what can be done in this direction cannot do better than read his lectures in the New Sydenham Society's translation, if not in the original. To him especially we owe our knowledge of the vascular changes which occur in inflammation, and which we shall discuss directly.

But first we must consider for a moment the "reaction to injury" in the lower animals. This has been very carefully worked out by Metchnikoff ('Pathologie comparée de l'Inflammation') and others.

Among the Protozoa simple mechanical injury is followed by immediate repair; thus if a large amœba be divided by a cut the separated parts heal up, and that part which contains the nucleus continues to grow and proliferate as before. But if the amœba receive what may be called a chemical injury the reaction is different; the structure of the protoplasm is affected, and an extrusion of hyaline globules may be brought about, resembling what is sometimes seen in cells of an inflammatory area, and possibly similar to the extrusion of granules described by Kanthack and Hardy in leucocytes in the presence of bacteria. Again, if foreign particles of matter be introduced into its neighbourhood, the amœba tends to take them up and to digest them in the acid-containing vacuole which it forms around the particle ingested. Here, then, we have already an evident distinction between the reaction to mechanical and other forms of irritation.

Coming next to the Metazoa, which have ectoderm and entoderm and mesodermal wandering cells, it is found that the reaction to injury and irritation is a reaction of the mesodermal cells, a group which has preserved a marked resemblance to the simplest protozoon forms of life. Thus Metchnikoff found in studying certain larvæ that if a sharp foreign body be pushed through the ectoderm the mesodermal cells collect and form a plasmodium round it, and if bacteria are introduced they are taken up and speedily digested by the wandering cells. And similar facts have been observed in worms.

In the crustaceans also the reaction to the introduction of foreign irritating matter, such as bacteria, is a reaction of their leucocytes. And Metchnikoff, in studying a disease of Daphnia (the water-flea) due to infection with the monosporon, showed that in this animal the spores are eaten up within the body by the leucocytes, and thus destroyed; but if too many gain access, and are thus enabled to develop into torulæ, the latter cause destruction of the leucocytes and subsequently bring about the death of the animal itself. The reaction of the leucocytes, then, is a protective one. This is well seen in the same animal—Daphnia—after an injury to its carapace. The first thing that occurs is the collection of a mass of leucocytes at the site of injury, where they destroy whatever foreign matter may have entered through the breach, and close the path of access against further irritating substances. Then follows a reaction of regeneration or repair, and only when the loss has been restored by the proliferation of the tissue-cells is the leucocytic barrier removed.

The reaction of repair is very extensive in the lower animals; thus worms replace their hinder segments after these have been destroyed, crustaceans can restore lost claws, and lizards will renew a severed tail. But this regeneration is quite distinct from the direct response to injury; it occurs much later, and is brought about by a proliferation of fixed tissue-cells in which both epiblast, hypoblast, and mesoblast may all take part. The reaction of regeneration cannot be regarded as being in any way a part of the inflammatory process in these animals. It is a subsequent but not essentially a consequent reaction.

If now we pass on to examine the events of in-

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flammation in the vertebrates, with which we are naturally more particularly concerned, we find that vascular phenomena almost invariably play a prominent and important part. It is to those phenomena that the heat, redness, and swelling which are usually associated with inflammation are due. They were first thoroughly investigated by Cohnheim in the web of the frog's foot, and nothing of essential importance has been added to his results by subsequent observers.

If the epithelium of the frog's web be slightly injured with a hot wire or by a drop of acid, and be watched continuously under the microscope, the following alterations in the circulation may be seen to occur :

1. Sometimes a very brief *constriction* of the arterioles of the part. This is probably the direct effect of the injury rather than a reaction to it.

2. *Dilatation* first of the arteries, later of the veins ; and in perhaps an hour a dilatation of the capillaries also.

3. *Acceleration* of the stream in the dilated vessels, the axial stream becoming very marked. This is followed by a gradual—

4. *Slowing of the stream* until the actual current, which has broadened out more and more, is ultimately lost. Meanwhile the leucocytes appear to have become "sticky," and tend to adhere to one another and to the vessel walls in the part affected. This process and the progressive slowing of the stream may end in complete—

5. *Stasis*, though it does not always nor necessarily do so. At the same time an—

6. *Exudation* of lymph is occurring from the vessels, and—

7. *Diapedesis* leading to the so-called "margination," of the smaller veins especially, with leucocytes, many of which gradually pass out to reach the injured surface, covering it eventually with a thick layer, which dries to form the scab.

Gradually the circulation recommences, and by degrees returns again to normal, exudation and diapedesis ceasing. Then within perhaps about twenty-four hours the process of regeneration commences, the epithelium at the edges of the wound begins to proliferate and spread beneath the scab, and healing rapidly proceeds. From such observations Cohnheim came to the conclusion that the main thing in inflammation is the succession of vascular changes, and formed the theory that this process was due to a molecular alteration in the vessel walls. It could not be dependent on the formation of new vessels as Hunter had supposed, for such formation does not necessarily occur, and where it does it is a late phenomenon.

The original experiment of Cohnheim has been modified and improved by various observers, and especially by Burdon Sanderson and Stricker, who investigated inflammation in the exposed omentum of mammalian animals, by Thomas, of Heidelberg, and by Pembrey in the mouse. Coats' modification of the frog-web experiment, in which he snipped away a small area of the epithelium with sharp scissors, had the great advantages that practically the only injury inflicted was exposure of the subcutaneous tissue to the air, and that the vessels in the tiny wound could be examined without the intervention of a dead epithelial covering.

But inflammation does not always terminate in resolution in the way I have described. It may go on to suppuration or necrosis. Suppuration is best seen in inflammations due to the action of micro-organisms, though, as we now know, it may also be induced by means of powerful chemical irritants. Here the process goes on beyond the stage of stasis, the vessels in the centre of the affected area become filled with leucocytic thrombi, and diapedesis proceeds until the area is densely packed with white corpuscles. Destruction of tissue and degeneration of leucocytes takes place in the central area, where the agent of injury or noxa is present in greatest concentration, resulting in the production of a purulent fluid—the liquor puris—while round the circumference is a layer of closely packed white corpuscles, and round this again a layer of rapidly proliferating connective-tissue cells, which form the so-called granulation tissue, itself infiltrated with leucocytes, and constituting the pyogenic membrane by which the contents of the abscess are encapsuled.

These changes we shall have to treat more fully later. Meanwhile, we must now consider briefly the phenomena of inflammation in non-vascular areas.

The most convenient site for studying this in higher animals is in the tissues of the cornea. And very numerous are the observations which have been recorded. The most complete, perhaps, are those of Senftleben, who was the first to use the cornea for this purpose. If the central part of the

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cornea be lightly touched with a strong solution of zinc chloride (60 per cent.) without causing any breach of the corneal epithelium, there is seen to follow a degeneration of the cells (corneal corpuscles) in the centre of the area affected by the caustic action. The cell-bodies disappear, and only traces of the nuclei remain. Around this central area is a zone in which the cells are shrivelled and coagulated. In about forty-eight hours the process of repair begins just like repair in ordinary connective tissue. The sound corpuscles at the margin of the injured part begin to throw out processes—"regeneration spikes," as they are called—which gradually grow into and eventually fill up the spaces of the injured area from which cells have disappeared, and from these "spikes" new corneal corpuscles develop. There is no change in the vessels at the margin of the cornea, and no invasion of leucocytes occurs; that is to say, regeneration has occurred without reaction of the leucocytes. Here there has been no inflammation, although the process is associated with the antecedent injury, and was initiated by the effects that injury produced.

If, now, the same experiment be done near the margin of the cornea—that is, nearer vessels—or if, a few hours after the central cauterisation, we inflict a tiny wound by lightly scratching off a fragment of the epithelium which was cauterised with the zinc chloride, there follows infiltration of the tissue-spaces with a mass of leucocytes which come in the former case out of the neighbouring vessels, and in the latter from the conjunctival fluid. And this gives rise to localised opacity, in fact, a local keratitis.

From these experiments we gain two very important conclusions:

1. That the keratitis—the inflammation—was dependent on a leucocytic immigration.

2. That the process of repair was not dependent on migrated leucocytes, but on the tissue-cells.

To proceed a little farther: Metchnikoff has shown that if the epithelium of the tail fin of a certain embryo (the axolotl) of about two weeks old, in which the tail fin is non-vascular, be slightly injured by touching it with silver nitrate, the following series of events occurs:—(*a*) A certain number of cells are immediately destroyed; others are injured, and these become swollen up by imbibition of water. (*b*) An immigration of leucocytes occurs; but these are wandering cells of the tissue, for there is no change in the nearest vessels, and no diapedesis of their white corpuscles. (*c*) Repair by proliferation of the injured epithelium.

In the tail of the newt a similar series of changes may be seen, but here there are vascular events associated. Stasis occurs in the nearest vascular loop, accompanied by diapedesis of the leucocytes. By the following day the parts have again returned to normal. In the newt the vessels are much more numerous and much nearer to the site of injury than in the axolotl, whose tail fin contains no blood-vessels. The newt experiment is therefore comparable to the experiment at the margin of the cornea.

From all these observations it follows that the view of Cohnheim, which regarded inflammation as being determined by molecular changes in the vessel walls, must be abandoned. It follows also that since regeneration may occur without the occurrence of any inflammation it cannot be regarded as a part of the inflammatory reaction. The restoration of the cornea, after central cauterisation unaccompanied by any breach of the epithelial covering, proceeds without inflammatory reaction; the same is true of injuries purely mechanical in nature. Accordingly we must regard the repair accompanied by or following an inflammatory reaction as being only an associated process and not an integral part of inflammation as at present understood. On the other hand, it may eventually prove that both these processes are only the expression of the same general reaction as exhibited by different cell-systems of the body.

We must now pass on to a consideration of the part played by the blood-vessels in the inflammatory process.

The heat, redness, and swelling of an inflamed part are all dependent on the local changes in the circulation which we have already described. Redness is due to increase of blood-supply and dilatation of the capillaries; and swelling, to an increased transudation from the vessels. The increased heat is due to increased volume and velocity of the local circulation, and to this alone. The temperature of inflamed tissues never exceeds that of the circulating blood. This was first taught by Hunter, as the result of experiments in which he found that the temperature of the rectum and vagina of animals was but little affected by

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acute inflammation artificially produced in those parts. It was proved by Cohnheim, who showed that the temperature of inflamed parts is never greater than that registered by a thermometer introduced into the rectum. If fever is associated with the inflammation, and the general blood-temperature is raised, the inflamed part will then be hotter than the normal; but it is never hotter than the blood itself.

There is therefore no production of heat in inflamed areas. On the other hand, the temperature of an inflamed area may be less than that of the body generally, and may even be subnormal. In artificial pleurisy, for example, the temperature of the inflamed pleura may be lower than that of the healthy one. This is due, when it occurs, to a diminished blood-supply resulting from the condition of complete or partial stasis in the vessels of the affected part, which succeeds the earlier stage of increased and accelerated current. And Cohnheim showed that if one fore-paw of a dog be caused to inflame, while the other is simply rendered hyperæmic by division of the brachial nerves upon that side (the arteries in consequence dilating), the latter has slightly the higher temperature. Because while the circulation in the inflamed paw becomes eventually slowed, that in the opposite limb remains accelerated. The observations of Cohnheim have been confirmed by Jacobsen in experiments and with instruments of extreme precision.

I have already described the succession of vascular changes which occur in inflammation; we must now consider briefly their causation.

The preliminary brief constriction of the arterioles is probably resultant, not reactive—the direct effect of the injurious agent, not a reaction to it;—and it is quickly followed by a reactive dilatation. This change in calibre of the arterioles is not the consequence of central reflexes, for, as I have stated previously, complete division of the nerve-trunks does not affect the succession of these changes; and Cohnheim showed that even when the vessels are already dilated after nerve section, a further dilatation follows if a local inflammation be induced in the infected area. The inflammatory dilatation must be either due to direct action of the irritant on the vessel-walls, or indirectly through the agency of the peripheral nerve-endings in those walls. Between these possibilities we cannot decide on present knowledge, but it is of suggestive interest to remember that stimulation of the peripheral portion of a mixed nerve often produces at first a brief constriction, followed by lasting dilatation of the vessels in its area of distribution.

The accelerated circulation is the direct result of the dilatation of the arterioles; since more blood necessarily flows to a part in which the arterioles are dilated than to the neighbouring parts supplied by the same artery; and therefore if the pressure in the main artery undergoes no diminution an increased amount of blood will reach the part whose vessels have become dilated.

The subsequent slowing of the circulation in the still dilated arteries might be attributed to any one of the three following factors: an alteration in the vessel walls causing increased resistance to the flow of blood, or an increased resistence due to the great massing of leucocytes in the small veins and also in the capillaries, or to a local increase in the specific gravity of the circulating fluid consequent on the enormous amount of transudation taking place. Probably all these factors play a part in the result. But that an alteration in the vessel walls is the chief factor follows from the experiments of Ryneck, who was able to show that stasis can be artificially produced in vessels through which an indifferent fluid, such as milk, is being circulated, but cannot be produced at all if the vitality of the endothelium be first destroyed by circulating a solution of a metallic poison.

That alterations do actually occur in the endothelial cells in inflammation is evidenced by the fact that they may be observed to enlarge, at times projecting into the lumen of the vessel, to undergo proliferation and multiply, to throw out pseudopodia, and in bacterial inflammation to take up the micro-organisms by phagocytic action. They appear also to become adhesive, so that the leucocytes and even red corpuscles now exhibit a tendency to stick to the vessel walls; they offer increased resistance to the circulation; and they permit the transudation of increased quantities of altered lymph. In chronic inflammations also, according to Arnold and most other recent observers, they give rise to the new vascular loops which form in the inflammatory tissue, though this has been denied by Rindfleisch, who attributes the origin of new capillaries to the tissue-cells.

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ON RECRUDESCENCE OF INVETERATE LATENT SYPHILIS AFTER ACUTE FEBRILE DISORDERS.

By HEINRICH OPPENHEIMER, M.D.Heidelberg, M.R.C.P.London.

I HAVE often been struck by the frequency with which acute febrile disorders are instrumental in lighting up the fuel left in the system by inveterate syphilis. And as I think the subject has not received the attention it deserves, I consider the following three typical cases, of which I possess complete notes, worth publishing.

CASE 1.—A man, æt. 49, having just recovered from a severe attack of influenza of the gastro-intestinal type, with a temperature running up to 103°F., consulted me on September 26th, 1899, for a painless enlargement of the right testicle, which the patient declared to have set in rather suddenly within the last week. The swelling was limited to the body of the organ, which was increased to about twice its normal size, globular in outline, homogeneous, hard, and not tender to the touch. The tumour possessing all the characteristics of a diffuse syphilitic orchitis, I asked the patient when he had acquired syphilis, but I met with a flat denial. Remembering that cases of orchitis had been recorded as occurring as a sequel to influenza, and being aware that the patient had been married more than twenty-five years and had had a family of healthy children, I was satisfied for the moment with considering the case as simple orchitis and with prescribing a non-specific treatment. A week later the patient presented himself again, and, on examination, I found that the swelling had increased rather than diminished, and that it was unchanged in its other characters. Without any further inquiry I now put the patient on iodide of potassium with the most satisfactory results, the enlargement completely disappearing in less than a fortnight. On being pressed the patient now confessed having had a syphilitic chancre at the age of nineteen; he was then sent to Aix-la-Chapelle, where he underwent a month's treatment, when he was pronounced cured. Apparently this opinion seemed to be justified; for though he never since took mercury in any form, he remained free from specific manifestations for a period of quite thirty years, married, and begat a healthy family; none of his children ever showing the least indication of an inherited taint, and his wife never aborted. What is further remarkable is the fact that the patient consulted me again on December 27th of the same year, this time for hoarseness, when the laryngoscopic examination revealed a well-marked gumma of the size of a split pea on the epiglottis. Iodide of potassium rapidly effected a cure, and the patient has had no symptom since.

CASE 2.—This patient was a man æt. 54, who, in March, 1900, had an attack of acute right lobar pneumonia. The disease took a normal course, but a day or two after defervescence had been established the patient commenced to complain of severe occipital headache of a neuralgic type. The ordinary nervine and antineuralgic remedies failed to give more than slight and temporary relief, and the pain persisted with varying severity till the beginning of May, when the patient came to see me for diplopia, which proved to result from a well-marked paralysis of the left rectus externus muscle. Iodide of potassium was prescribed. After the third or fourth dose, the neuralgia subsided, and has not been complained of since. The ocular symptoms likewise soon began to improve, and by the end of June the patient was quite cured. This patient had been infected when seventeen years of age, had mild secondary symptoms during the first few months, and took mercury internally for about six months altogether. Since then he had kept well, had married at the age of twenty-nine, and two years later a healthy child was born to him, a daughter, now alive and well. I may add that the knee-jerk was normal at the time of the ocular palsy, and is so now.

CASE 3.—This patient, a man æt. 46, suffered from an attack of rheumatic fever of moderate severity and of six weeks duration in September and October, 1901. During convalescence, which was somewhat protracted, though no complications arose, the patient got hoarse and began to complain of an irritating spasmodic cough. On laryngoscopic examination I found a diffuse infiltration of the epiglottis and of the posterior laryngeal wall, with a few isolated slight gummous

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protuberances in the latter locality. These deposits quickly disappeared under the administration of potassium iodide. The patient had contracted syphilis at the age of twenty-five, a slight early secondary rash being the only secondary manifestation. A six weeks' course at Aix at the time was the only mercurial treatment he ever underwent. He had kept well up to now; married at the age of thirty-seven, but there is no issue of the marriage, and his wife never had a miscarriage.

It is a well-known fact that under the influence of weakening factors of any kind relapses are very liable to occur. But the experience in these and other cases has convinced me that febrile disorders are more frequently than other causes instrumental in imparting fresh activity to syphilitic residues in the system which have been in a dormant condition for many years. These unexpected manifestations do not break out on the height of the fever, but belong to the stage of convalescence, and to the period immediately following the latter, when the patient, to all external appearances, is quite well again. Cases of a mild and abortive type seem to suffer most frequently in this peculiar way, probably on account of the insufficient and unsatisfactory treatment with which they are too often content.

Are not some Deaths during Operation in the Region supplied by the Trifacial Nerve due to Reflex Inhibition of Respiration and of the Heart?—William Harmar Good says it is a well-known fact that the peripheral sensory nerves all stand in a reflex relation to the cardio-inhibitory centre, and also that the respiratory centre is readily excited through reflex action, as instanced by the effect of cold water suddenly dashed upon the body, or by the accidental inhalation of strong ammonia. In etherising rabbits, it is noticed that as soon as the ether is applied to the nose the animal stops breathing for a more or less prolonged period. At the same time the heart is markedly inhibited; in fact, almost stopped. The recognition of these facts gave rise to the following questions:—(1) Is the inhibition due to the effect of the ether directly on the respiratory and cardio-inhibitory centres? (2) Is it due to irritation of the vagi-endings in the lungs? (3) Is it due to irritation of trifacial fibres in the nasal mucosa, and if so, would other irritants act similarly? The writer answers these questions by pointing out that in the etherisation of rabbits the reaction is so rapid as to preclude the possibility of the absorption and transmission of the ether through the blood of the medulla. Also that the application of ammonia or pinching the tip of the nose has the same effect. And further, that from experiments on rabbits it can readily be proved that the respiratory and cardiac movements can be reflexly inhibited by irritation—either mechanical or chemical—of trifacial nerve-endings. This raises the question whether, during general anæsthesia in man, irritation of the trifacial nerve may not cause a reflex inhibition of respiration and of the heart, and death ensue? This result would occur most likely during partial or incomplete anæsthesia. After citing a few cases tending to prove the theory advanced, the author suggests methods of prevention and of overcoming the reflex after it has taken place. He points out that, granting reflex inhibitions, it follows that the state of primary anæsthesia is a dangerous time to perform any operation. It should not be commenced until anæsthesia is complete and the conjunctival reflex has been abolished. Moreover the patient should not be allowed to come partially from under the influence of the anæsthethic during the operation. Finally, he emphasises the treatment advanced theoretically by Ott and practically proved by Jackson. It consists simply of distension of the lungs by forced inspiration, by mouth-to-mouth insufflation, or even by means of the bellows, as is done in experimental work on animals.—*Medical Record, August 30th, 1902.*

Medical Treatment of Tuberculous Peritonitis.—Persistence in the local application of equal parts of iodoform ointment and cod-liver oil is of extreme importance. A mixture of 15 gr. bismuth salicylate, 15 mm. spr. chloroform, 3 ½ comp. tinct. cardamom, with mucilage and water will relieve pain and allay intestinal irritation.—*Med. Record.*

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THE VALUE OF TENDON-JERKS AND SUPERFICIAL REFLEXES IN DIAGNOSIS.

A Clinical Lecture delivered at the National Hospital for the Paralysed and Epileptic, Queen Square, May 13th, 1902.

By J. S. RISIEN RUSSELL, M.D., F.R.C.P.,

Physician to Out-patients at the Hospital.

GENTLEMEN,—The subject which we have chosen for this afternoon's lecture is the value of the tendon-jerks and superficial reflexes in diagnosis. The subject is a very large one, and I cannot possibly pretend to treat it exhaustively in the time at my disposal for one lecture. So I propose to confine myself to certain points which may be more or less illustrated by cases which we happen to have in the hospital. I shall limit myself to four main considerations. Firstly, the value of the tendon-jerks in the differential diagnosis between organic and functional affections of the nervous system; secondly, the value of these phenomena in the differentiation of one organic affection from another; thirdly, their value in the localisation of disease in the spinal cord; and, lastly, though I am afraid time will not permit of it, I should like, if possible, to show their value in regard to prognosis in disease of the spinal cord. You see, then, that I shall not try to go exhaustively through the whole subject, but intend to limit myself in the way I have indicated.

The first thing I wish to deal with is the state of the tendon-jerks in cases of functional affections of the nervous system, and the first patient I shall bring in is to illustrate this. The patient is suffering from neurasthenia. In neurasthenia you may meet with two conditions of the tendon-jerks; one is exaggeration and the other is diminution of them. Now, it is infinitely more common to meet with exaggeration of the tendon-jerks in neurasthenia than to meet with anything like diminution of them.

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So much is this the case that, while I am able this afternoon to show you a case of neurasthenia with exaggerated jerks, I am unable to bring before you a case of neurasthenia with diminished tendon-jerks. Nevertheless, that such a state of things does exist in that affection is well recognised. Indeed, I have seen cases of neurasthenia where the knee-jerks have been said to be absent, but where in reality they were greatly diminished, but could be brought out by reinforcement. So that great diminution may occur, but much more commonly you will find increase of the jerks. This first patient will serve to illustrate the fact that the knee-jerks are excessive both on the left and on the right side. In spite of this great activity of the knee-jerks, I call your attention to the fact that there is no ankle-clonus. There are a few little jerks at the beginning, when one attempts to elicit clonus, a kind of pseudo-clonus, but it does not persist. These slight jerks sometimes lead to the supposition that a real ankle-clonus exists when it does not. There is no ankle-clonus of a genuine kind here. Further, on stimulation of the soles of the feet we find that there is nothing suggestive of the alteration of the plantar reflex that we meet with in organic disease, and which is known as Babinski's sign. What we get here is merely a slight flexion of the toes, the normal form of response. I will not waste your time by examining the other limb, but I will ask you to believe me that what we have been able to demonstrate on the right side exists also on the left, and that with these excessive knee-jerks there is no evidence of any ankle-clonus, and that plantar stimulation does not elicit the extensor type of response met with in organic affections of the pyramidal system. So, too, the arm-jerks are increased in many of these cases of neurasthenia. I will illustrate this by eliciting the supinator jerk, which is increased in this woman. We see, therefore, that the tendon-jerks in the upper and lower limbs may be excessive in a functional affection such as neurasthenia, without there being any evidence of organic affection. It is important for you to recognise these points, because from time to time one sees cases of this kind where the diagnosis of lateral sclerosis is made, the increased activity of the jerks being regarded as dependent upon lateral sclerosis in the spinal cord. Of course the prognosis is influenced in a very serious way by a diagnosis of that kind as opposed to a correct diagnosis of the real condition existing, namely, a functional disease of the nervous system which can be recovered from. I am sorry I cannot bring before you a case illustrating diminution of tendon reflexes in neurasthenia, but there is no doubt that there are such cases.

The next patient I will show you is in order to illustrate that similar exaggeration of the tendon-jerks may exist in another functional affection of the nervous system, namely, hysteria. This patient has motor defect mainly in connection with one limb, but when she walks you will see there is some defect on both sides. Notice the stiff way in which she walks, and the tendency to cross-legged progression which she exhibits. In her we can find no evidence whatever of organic affection. Nevertheless, her knee-jerk is increased on the right side, and similarly it is increased on the left. But in spite of the increased knee-jerks we have no sign of ankle-clonus, and no sign of the plantar reflexes being altered in the way that we meet with in organic affections. There is no sign of the extensor response on stimulation of the sole of the foot. She does not illustrate what might have been met with in a case of this kind, viz. a point to which Dr. Buzzard has drawn attention in relation to the question of the state of the plantar reflex in functional paraplegia. Dr. Buzzard called attention to the fact that although the knee-jerks are so often exaggerated in these cases, it is no uncommon thing to find that the plantar reflexes are absent. In this girl the plantar reflexes are preserved, but in many cases of functional paraplegia you will find that on stimulation of the soles of the feet you get no response at all in so far as the toes are concerned; they remain immobile. The case illustrates what we usually find in regard to the tendon-jerks in cases of hysteria. It requires great boldness on the part of any individual to diagnose hysteria in a case in which the tendon-jerks are absent. The plantar reflexes may be absent in a case of functional disease, hysteria, but the knee-jerks, as I have shown you, are increased: and that is the rule. It would, I say, require great boldness on the part of anybody to diagnose a functional condition, such as hysteria, when the knee-jerks are absent. I remember a case where this was well illustrated in a young lady who was afterwards operated upon in this hospital. She

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first came under my observation in another hospital. She was being at that time, and had been for more than a year, treated for functional disease, that is to say, functional paraplegia. Her first manifestations had been a slight dragging of one leg, the right one. And then she got some difficulty in walking, both legs being affected, and finally, for two months before I saw her, she had not been able to stand or walk; she had been treated as a case of functional paraplegia, hysterical. Without going into other points in connection with that case, I may say that the first thing that arrested one's attention, and that negatived absolutely the idea that we were dealing with a functional paraplegia, was the fact that her right knee-jerk was absent. So one was able to demonstrate to those with whom one saw the case in consultation that the absence of the right knee-jerk excluded the possibility of a functional affection in that case. The case afterwards, as I have told you, was explored in this hospital, and sarcoma was found as the cause of the paraplegia; it was a compression paraplegia, due to sarcoma. That case, then, was one of organic affection which had been regarded and treated as functional, and in which this one sign alone sufficed in one's opinion to show it was not a functional condition, and to make organic disease certain. But, gentlemen, I would next quote a case in which absence of knee-jerk was the state of affairs, and yet one diagnosed functional disease. I was asked to see a young lady, also at the present time in the other hospital to which I have just referred. The history was that for ten years she had been suffering from sciatica—the first manifestation had been pain in connection with the left leg; that the sciatica had been extremely chronic; and that she had seen a great many people about it. Finally, the condition appears to have been regarded as a rheumatic condition of the hip and knee, and she was sent to Bath, where she underwent treatment without benefit. For two years before I saw her she had been practically confined to bed; part of the day, however, was passed on a spinal couch. The condition of things present was that there was extreme atrophy of the left lower limb, and atrophy of the right lower limb also, but not to anything like the same extent as the left; and with this extreme atrophy of limb there was marked contracture. The limb was drawn up and fixed at the knee and hip. The knee-jerk could not be obtained on the left side. The right knee-jerk, however, was present; indeed, it was active. In view of the fact that the left knee-jerk could not be obtained, one hesitated in the diagnosis which otherwise seemed fairly obvious, namely, that it was a case of hysterical sciatica, with these changes secondarily occurring from disuse and from immobility of the limb. We placed her under an anæsthetic, and broke down the adhesions, and yet at the stage of chloroform narcosis, in which one would have naturally expected to have elicited the knee-jerk, the knee-jerk was still in abeyance. Under treatment, however, this girl is now able to walk, and, moreover, her knee-jerk has returned, so that it is now practically as well marked on that side as on the other. In this case there were reasons why the knee-jerk might be absent. There was fixation of joints, notably the knee-joint, and there was the marked atrophy of the muscles of the limb. In the other case which I quoted before there was no such condition present which would explain the absence of the knee-jerk in that way; the limb was not atrophied, there was no fixation of the knee-joint to account for the knee-jerk being absent. I have quoted these two cases in conjunction to show you how, under certain conditions, it may be necessary to arrive at the diagnosis of functional disease in a case in which the knee-jerk is absent, while in another case the absence of the knee-jerk may be taken as positive proof of organic disease. But I wish to strongly emphasise the fact that this should be a very rare event, as there are few cases which are likely to call for such a diagnosis under these circumstances, and that without an adequate explanation for the absence of knee-jerk in any given case, the diagnosis of functional disease is not admissible, as organic disease probably exists in a case of that kind.

I want to bring this next patient before you in order to illustrate the point which I have just referred to in connection with the case of the girl with absent knee-jerk, in whom one was obliged to arrive at the diagnosis of functional disease in spite of the knee-jerk being absent. I said that in her case there was an adequate explanation in the fixation of the joint and the contracture which was present. Now, here is a woman with unquestionable evidences of organic disease of her spinal cord, and it is of a character which would lead us to

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expect exaggeration of her knee-jerks and the ordinary phenomena otherwise met with in such cases, namely, ankle-clonus, extensor response, and so on. As a matter of fact, there is great contracture here, which one can only partially overcome, and you will observe that we are unable to get the knee-jerk. I may tell you that Dr. Barnes, the senior house physician, has been carefully over the case, and has been unable to elicit the knee-jerk. Now, in spite of the absence of the knee-jerk in this case, the extensor response has been obtained. The patient has a lesion of her spinal cord in which we should not expect her knee-jerks to be abolished, in that the position of the lesion is too high; and that it is not a complete transverse lesion is evident from the degree of spasticity present. This condition of contracture, then, probably accounts for the fact that we cannot obtain her knee-jerk. But although her knee-jerk is absent the plantar reflex is present, and gives the abnormal response, namely, extension of the great toe, especially on the left side. On the right side the response is not so brisk. In the case I quoted, of the young lady with hysterical manifestations in connection with the left limb in whom the knee-jerk was absent, that sign was not accompanied by any other signs of organic disease, such as the extensor response, which we have demonstrated in the case which you have just seen.

The next case we wish to show is another instance of an hysterical affection, namely, hysterical hemiplegia. One brings the case before you because it illustrates the need for a good deal of courage in the diagnosis of hysteria under the circumstances. For this man has the most profound atrophy of his limbs on the affected side. The hemiplegia has existed for five years, and, as a consequence of the disuse during this long time, we assume that this profound wasting on the right side has resulted. The same difference may be noticed in his lower limbs. The hemiplegia which exists in this man is of the absolutely flaccid type; his limbs on the right side are quite flail-like, and there is no sign whatever of spasticity. But in spite of that you will find that the knee-jerk is present on both sides, and that it is more active on the right side than on the left. No ankle-clonus can be obtained, and no plantar reflex has been elicited on the right side; on the left side there is the normal flexor response. Here, then, is a case in which we regard the hemiplegia as functional; but there is nevertheless marked muscular atrophy in these limbs; and in the presence of such marked muscular atrophy one would naturally be inclined to diagnose an organic affection. But so far as the tendon-jerks are concerned, they point to the condition being functional, and the plantar reflex which we obtain supports the diagnosis further. In organic hemiplegia you would expect some spastic element in connection with the limbs—increase of the jerks, such as is present here; and in addition you may get ankle-clonus and the extensor response as the plantar reflex on the affected side. I need say little to you on this subject, however, as the diagnosis between functional and organic hemiplegia was considered last week when Dr. Buzzard lectured to you. All I wish to emphasise is that the knee-jerk is increased; it is more active on the affected side than on the other, and yet there are no other signs indicating the presence of an organic affection. There is complete absence of spasticity, there is no ankle-clonus, and the special form of alteration of the plantar reflex met with in organic affections of the pyramidal system is not present.

The next patient I wish to bring before you is one of ordinary hemiplegia. I bring the case immediately after the case of functional hemiplegia, because it does not conform to the complete picture of a hemiplegia such as you might get in regard to the alterations of the tendon-jerks. He is suffering from slight left hemiplegia. To remove any doubt which any one may have as to its being a case of hemiplegia, one might demonstrate the fact before going on to the main points. His grasp is weaker on the left side than on the right, and the power in his left leg is not as good as that in the right. He has, then, hemiplegia, and it is of organic origin; thrombosis being the probable lesion. His arm-jerks on the right side are slightly in excess of those on the left. His knee-jerk is distinctly increased on the left side, as compared with the right. But although we are sure we are dealing here with a case of organic hemiplegia, he no more illustrates the other signs which we expect in an organic hemiplegia than did the man who has just left the room, in that there is no sign of ankle-clonus in the case, there is some spasticity, and one has been unable to

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obtain his plantar reflex so far. His foot is rather cold, and of course it would not be fair to say that it absolutely is not there until one tried with the warm feet in bed. But in this man with a left hemiplegia, and other evidences pointing to the hemiplegia being organic, there are no more the signs which we expect in an organic hemiplegia, as far as tendon-jerks and superficial reflexes are concerned, than in the other man. Stimulation of the sole of the foot does not elicit the reflex on either side. I bring him in not as an illustration of what we get as a rule in organic lesions, but to emphasise the fact that diagnosis is not so easy in these cases as is sometimes thought, and that while the tendon-jerks do help us in the great majority of cases, you must expect to meet with exceptions, and this man is a good illustration of an exception to the rule.

This next patient, a woman, is also a case of hemiplegia, and her hemiplegia is on the right side, associated with some aphasia. She, like the man you have seen, presents an exaggerated jerk on the affected side, There is an increase of her knee-jerk, no ankle-clonus on the left side, but she has some ankle clonus on the right side. With regard to her plantar reflex, it is very distinctly of the abnormal type that we expect in organic hemiplegia, namely, extension of the great toe. That phenomenon existed in conjunction with the exaggerated knee-jerk before, but she did not present ankle-clonus, although she does so now. In short, she now presents the ordinary features one would expect in organic hemiplegia. The other case, in a man with equally definite organic disease, does not conform to the rule; he proves a notable exception in that he has neither ankle-clonus nor extensor response, and yet we know by other signs that we are dealing with a case of organic hemiplegia.

So much for those few points in regard to the value of the tendon-jerks and superficial reflexes in regard to the differential diagnosis between organic and functional disease. But I cannot leave this part of the subject without referring briefly to a sign to which attention has been called more or less recently, namely, the value of the tendo-Achillis jerk in the differential diagnosis between functional and organic sciatica. In cases of sciatica, notably in women, as you are aware, it is oftentimes difficult to be sure whether we are dealing with a functional condition, or whether we are dealing with the real disease. According to Babinski, who is responsible for this information also, the tendo-Achillis jerk is commonly absent on the affected side in a case of organic sciatica, whereas it is preserved on the affected side in hysterical sciatica. The tendo-Achillis jerk, then, is lost on the affected side in organic sciatica, and it is present on the affected side in hysterical sciatica. Now I want to give you a word of caution with regard to the tendo-Achillis jerk: be sure that you get this jerk on the normal side before you draw any conclusions from what you find on the abnormal side. For you will find that there are many cases of patients where it is difficult to obtain the tendo-Achillis jerk at all definitely, and in which, therefore, the jerk may appear to be absent, and you may conclude that you are dealing with some organic affection, owing to the absence of that jerk, whereas in reality it is a condition natural to the individual whom you happen to be examining.

The next cases which one wishes to deal with are those in which the condition of the tendon-jerks prove of value in the differentiation of one organic affection from another. I said originally we would try first to deal with the value of tendon-jerks and superficial reflexes in the differentiation of organic disease from functional affection of the nervous system, and that we would next deal briefly with their value in the differentiation of one organic affection from another. The first cases I want to call your attention to are two patients, a brother and sister, suffering from myopathy. In them there exists a combination by atrophy of certain muscles and enlargement of others, this state of things being due to changes in the muscles which do not depend on any lesions that have been discovered in the central or peripheral nervous system adequate to account for the abnormal state of the muscles, so that the affection is regarded as one in which the muscles are primarily at fault. The main features which these two cases present are the following:—Muscles about the proximal segments of the limbs and certain muscles of the trunk are atrophied, while the muscles of the calves are enlarged, and, with the exception of the supinator longus, the muscles of the forearms have escaped, unless we admit some pseudo-hypertrophy of some of the flexor muscles

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of the forearm in the boy. The muscles of the hands have also escaped. In the case of the boy the hands and feet are short and broad, and they are blue in colour; moreover, the feet present the deformity known as *pes cavus*. The girl, as you can see, experiences great difficulty in rising from the sitting to the erect posture, and weakness of her trunk muscles is responsible for the marked lordosis of the spinal column in the lumbar region, which is so evident with her clothes on that there is no need for me to have her undressed to demonstrate this feature of the case. Now the tendon-jerks in both of these cases conform to the rule as to what we usually find in cases of myopathy, for the knee-jerks are absent, and there is no ankle-clonus. The knee-jerks are, however, not always absent in cases of myopathy. The absence of the knee-jerks in such cases depends on atrophy of the quadriceps extensor muscles, notably on the degree of atrophy of the vastus internus, so that when these muscles are not atrophied the knee-jerks are preserved. In marked contrast to the myelopathies, however,—that is, the muscular atrophies due to disease of the spinal cord,—the tendon-jerks are never increased, so that the knee-jerks are never exaggerated and ankle-clonus is never present. Moreover, the plantar reflex further serves to distinguish these cases from cases of muscular atrophy of spinal-cord origin, for while in the latter class the extensor response or Babinski's sign may be present, in cases such as those before us now this is never the case. In them the plantar reflex may be absent, as is the case in this boy to-day, but if present it is always of the normal flexor type, that is to say, the toes flex when the sole of the foot is stimulated; the toes never extend, as may occur when a spinal cord affection accounts for the muscular atrophy.

In marked contrast to the cases I have just shown you, in so far as the state of the tendon-jerks and superficial reflexes is concerned, I now wish you to see a case of muscular atrophy of spinal-cord origin. This woman is suffering from the disease known as amyotrophic lateral sclerosis, that is, the form of progressive muscular atrophy in which, in consequence of affection of the pyramidal system (upper motor neurons) in conjunction with affection of the anterior horn-cells (lower motor neurons) a spastic condition of the limbs exists in conjunction with muscular atrophy. In this case, in contrast to the other cases of muscular atrophy you have just seen, the muscles of the hands and those of the forearms are those that are markedly atrophied. Now, in spite of this pronounced atrophy of the muscles of the arms, the arm-jerks are not only preserved, but they are unduly active. So, too, she has an active jaw-jerk, and when we next examine her lower limbs we find that her knee-jerks are exaggerated, that she has ankle-clonus on both sides, and that her plantar reflex is of the extensor type, stimulation of the soles of her feet resulting in extension of the great toes, instead of the toes flexing in the way they do under normal conditions. The increased activity by the tendon-jerks, the presence of ankle-clonus, and the fact that the plantar reflex is of this abnormal type combine to make it certain that we are in the presence of a case of muscular atrophy of spinal-cord origin, as opposed to one in which the disease has originated primarily in the muscles without there being any disease of the spinal cord to account for the muscular atrophy.

Let us next consider how the tendon-jerks may help us to distinguish one form of disease of the spinal cord in which there is atrophy of muscles from another form of disease of the cord in which there is similar muscular atrophy. Here is a young woman in whom there is atrophy of the muscles of the left hand, with the characteristic deformity, *main en griffe*, which was also present in the last case you saw. There is also evidence of atrophy of the muscles on the ulnar side of the forearm. On the right side there are but slight signs of similar atrophy of the muscles of the hand. The case is one of syringomyelia, an affection in which, as you know, a cavity exists in the spinal cord. The most common seat of the cavity is the cervical region of the cord, and its implication of the anterior horns in this region accounts for the atrophy of muscles in the upper limbs. In contrast to the case of amyotrophic lateral sclerosis you have just seen, this patient's tendon-jerks are normal. The arm-jerks and knee-jerks are not unduly active, ankle-clonus is not present, and the plantar reflex is not altered in the way that it was in the last case. Matters might have been otherwise, however, for just as the cavity in the cervical cord may involve the anterior horns and occasion atrophy of muscles in the upper limbs in these cases, so it may involve the lateral columns of the

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cord, and thus lead to exaggeration of the knee-jerks, ankle-clonus, and the extensor response. In the case of the arm-jerks, however, this girl presents what is usually met with in syringomyelia, for in this affection the arm-jerks are usually normal or diminished, and not increased, as is the rule in amyotrophic lateral sclerosis. The next patient I wish to show you illustrates the fact that this rule has its exceptions, for this man is also suffering from syringomyelia, and yet observe that his arm-jerks are unduly active. Wherein does the difference between the two cases lie? In the position of the lesion; for whereas the girl presented no evidence of involvement of parts above the level of the cervical region of the spinal cord, the evidences in this man point to implication by the lesion of parts higher up, for he has paralysis of ocular muscles, of the muscles of one side of his face, of his tongue, half of which is atrophied, and so on. Thus it comes about that his pyramidal tracts have been invaded above the cervical region of the cord, and in consequence of this the arm-jerks are increased in activity. Indeed, the pyramidal fibres are implicated sufficiently high up to occasion even an active jaw-jerk in his case. You thus see that while in many cases the tendon-jerks help in distinguishing syringomyelia from amyotrophic lateral sclerosis, there are other cases, as in this man, in which they may afford us no assistance in attempts to solve the problem under consideration.

If you will allow me a few minutes longer, I am anxious to show you how the tendon-jerks and superficial reflexes may help us to localise the position of a lesion in the spinal cord. This man has caries of the spinal column and paraplegia as a result of compression of the cord. Now his knee-jerks are absent, and yet he has ankle-clonus, and the plantar reflex is of the extensor type met with in affections of the pyramidal system. In that the knee-jerks depend on the integrity of the the third and fourth lumbar segments of the cord, ankle-clonus on the fifth lumbar segment, and the plantar reflex on the first sacral segment, the particular combination present in this case, viz. knee-jerks absent, while ankle-clonus and the plantar reflex are present, means that the lesion must have involved the third and fourth lumbar segments of the cord, while the fifth lumbar and first sacral segments have escaped. As a matter of fact, this man is recovering, and one of his knee-jerks has in consequence returned. This is the first time that the knee-jerk has been obtained, however, for even when testing him to-day before the lecture we were unable to elicit the knee-jerk which you now see to be present. Some of you may say, But is it not a fact that in complete transverse lesions of the spinal cord above the lumbar enlargements the knee-jerks are absent? My reply must, of course, be in the affirmative. How, then, does it come about that I have committed myself to the assertion that in this man the lesion must have involved the third and fourth lumbar segments of the cord? Why is the case not one of complete transverse lesion of the spinal cord above the lumbar enlargement? Because ankle-clonus is present. The presence of ankle-clonus in this case while the knee-jerks are absent makes it certain that the lesion is in the position I have already indicated, unless it be that there is some mechanical impediment which prevents the knee-jerks from being elicited, as was the case in the woman I showed you when we had under consideration the differentiation of functional from organic affections of the nervous system.

In conclusion, let me show you a case which illustrates what we ordinarily find when we are dealing with an incomplete transverse lesion of the spinal cord above the lumbar enlargement. This man has spastic paraplegia consequent on an attack of myelitis of the thoracic cord many years ago. His knee-jerks are greatly exaggerated, ankle-clonus is marked on both sides, and he presents the extensor response in most typical form as the plantar reflex. This, then, is what we find in incomplete transverse lesions of the spinal cord above the lumbar enlargement, as opposed to complete flaccidity of the lower limbs with absence of the knee-jerks and no ankle-clonus in cases in which there is a complete transverse lesion.

Finally, let me say that in such transverse lesions of the cord as long as the tendon-jerks in the lower limbs are preserved there is a chance of recovery of motor power, whereas when the tendon-jerks are persistently absent in such cases there is no possibility of recovery. You thus see that the tendon-jerks may serve as an aid in prognosis as well as in diagnosis and localisation of diseases of the spinal cord.

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WITH SIR WILLIAM BENNETT
IN THE WARDS OF
ST. GEORGE'S HOSPITAL.

A Case of Varix of the Lower Limb.

You will perhaps recollect that on a former occasion I showed you a case of what was said to be varicose veins in the lower limbs, and I told you that no operation was indicated, as it was more likely to be productive of harm than good. The patient I now show has a condition of veins which affords an admirable contrast to that in the other case. The patient in the former case was a girl in the Winchester Ward, a very delicate anæmic subject, who had swollen legs on both sides, scattered over which was an almost uniform network of small veins, no one of the veins being larger than a piece of ordinary thread. Here and there were stellate vein areas, but there were no winding dilated veins which deserved the name of varix. She had been sent up for operation, but, as I have just said, I declined to do this because the veins were not of a kind which operation would, in my opinion, benefit; and especially because the swelling was not due to the condition of the veins, but was attributable to the anæmic condition of the patient at the time. Hers was, in fact, a condition which could be treated better by medicinal and hygienic means than by operation. Now here is a case of varicose veins in which operation is distinctly indicated. The patient, as you see, is a spare young man about nineteen years of age, whose occupation is that of footman; he is therefore very much on his legs. He finds that some veins which he has noticed to be large for a long time are beginning to give trouble. Towards the latter part of the day he has always felt recently a certain amount of aching in the legs, not in the thighs, and this aching affects on each side the middle of the back of the calf and the inner side of the leg as high as the knee. You will find that most of the people who complain of pains in connection with varicose veins in the leg will say the pain is in one of two places, or sometimes in both. One of these situations is a narrow area passing down the middle of the back of the calf, and the other is along the inner side and front of the leg. This pain may, as I have said, be localised in one of these two places, or it may affect both. There is, as you see, no swelling in the case before you. There are two distinct inconveniences which arise from varix of the lower limb. One results from the swelling, with or without œdema; the other is a localised pain, such as this patient feels. The pain in these two conditions is altogether different in origin, and the discomfort is of a different kind. On both sides you will note that this patient has well-marked varicose veins extending up the middle of the thigh from the foot. They are very obvious on the inside of each limb, and they are also pronounced on the back of the calf. The calf itself feels full and rather tense. The altered veins, as far as can be seen, are limited to the lower third of the thigh and the leg on each side. In point of fact, on one side it is only by careful palpation that the enlarged veins can be traced up to the thigh at all. The principal cause of the pain which this young man feels is irritation of the saphenous nerves; his discomfort is really due to a neuralgia of the long and short saphenous nerves. The cause of the pain in a case of this sort is not only interesting in itself, but very important from the point of view of treatment. Please note the absence of anything like œdema in this man's case. When he rises from his bed in the morning he is for all practical purposes well, but as the day goes on, and he is standing on his legs for long periods continuously, these become very full and tense, not from œdema, but from the increased bulk arising from the steady engorgement of the veins, especially the two saphenas. The result of this change is stretching and irritation of the saphenous nerves, producing the characteristic pain running along the course of these nerves. I need not, I suppose, remind you that in the leg the long and short saphenous nerves cling very closely to the corresponding veins. The pain, therefore, in this case is neuralgia, arising from the cause I have mentioned. The pain felt in cases in which there is œdema is of a different kind, consisting, mainly, of a feeling of general weight and stiffness, which only amounts to acute pain when the œdema is marked and unmistakable.

I now come to the question of the treatment of this case. It is clear that it is a case in which the veins are varicose, the cause of the pain being quite distinct; and it is, I believe, curable by

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operation. And in order to effect a cure no extensive operation is necessary if the proper proceeding be used. Let me tell you at once that an operation upon this man's veins *in the leg only* would be useless, for although *for a short time* it might seem to improve the condition of things, no real benefit would be derived from the treatment. The indication is to operate in the thigh upon the internal saphena as high up as possible. Please bear in mind that no good whatever will arise from any operation for varicose veins in the lower limb, small congenital varicose areas excepted, unless a short portion of the internal saphena vein be excised high up—the nearer the saphenous opening the better.

I was the first to point out the necessity of this measure, and practised it in this hospital as far back as 1883; a reference to it will be found in my lectures on varicose veins published in 1899. In these cases no operation will effect any useful purpose which does not include the removal of a portion of the saphena vein above the point at which it is joined by its posterior femoral branch, which enters it approximately about three to four inches below the saphenous opening.

There is a further point in this patient's case which opens up another question. Although, as I have said, the obvious varix is limited to the parts which we have seen, if you examine the saphenous openings carefully you will see that there is a saphenous varix on the left side; that is to say, there is at the saphenous opening a well-marked bulging of the vein, in which, upon a cough from the patient, a very distinct impulse with thrill can be felt. This dilatation affects partly the saphena and partly the femoral vein. The operation to which I have referred will not only relieve the discomfort arising from the varicose veins in the leg, but will be followed by the complete disappearance of the dilatation at the saphenous opening, a very important matter. In the majority of cases like that of this young man, the high operation is sufficient for the relief of all the symptoms from which the patient suffers, and excepting special cases, no operation upon the varicose condition in the leg is *necessary so far as the relief of the symptoms is concerned.* It is, however, *desirable* when the varix in the leg is very obvious, especially when well-marked pouches exist, to remove the more prominent portions. Such a proceeding is satisfying to the patient, as it effects an immediate and obvious alteration in the local condition. In the case of operations upon varix, for the purpose of obtaining admission to the public services, it is essential that the main obvious varicose veins should be removed. The main point, however, which I wish to insist upon, as I have so often before done, is this: that however obvious the varicose condition in the leg may be, no real cure or relief will be effected by operations confined to the leg, the only operation which will be effectual being the removal of a portion of the saphena vein below the saphenous opening, or, at all events, above the middle of the thigh, *i. e.* above the point at which certain tributary veins join the trunk of the saphena from the front and back of the thigh. Exception to this rule of treatment may be made only in certain cases of purely localised congenital masses of varix in the calf, which approach in character to coarse nævi. With regard to the after-treatment of these cases, you must first of all keep your patient on his back for at least three weeks or a month; and, secondly, when he gets up the vessels must be supported whilst the finality in the change in the circulation is coming about by an elastic support of some kind, such as an elastic stocking or bandage. It is essential to bear this in mind, because a belief is entertained by some people that once the veins are interrupted or removed nothing further is necessary, and that the patient may, at the end of a fortnight, get about and resume his business. But when this rapid plan is adopted these cases are almost always disappointing, with the result that the operation—which really is an excellent one when performed in the right way, and especially when the patient is afterwards managed in the right way—gets into bad repute. The great difficulty in private practice, and also to some extent in the hospital, is to get the patient in these cases to lay-up sufficiently long to reap the full benefit of the operation. If you interrupt by operation great venous channels like these are, in one or two positions, it is obvious that a considerable time must elapse before the vascular apparatus can accommodate itself to the altered conditions sufficiently to allow the circulation to go on properly when the patient resumes the upright position. As I have said, at least a month in the recumbent position is necessary if you are

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to obtain with certainty a good result in these cases, and then an elastic stocking should be worn for another three or six months at least.

A Case of Injury to the Hip.

Here is a woman who was knocked down by a cab, and fell upon her left hip heavily. On being brought to the hospital yesterday, she complained that she could not use the hip, on account of the pain which any attempt at movement caused. The position of the limb upon her arrival was practically as you see it now. It is, you will notice, apparently shortened somewhat, and is, in fact, just in the ordinary position which obtains in cases of intra-capsular fracture of the thigh bone, excepting that the eversion is very slight indeed. I am told by my house-surgeon, and I have no doubt it was so, that when she came in it was practically impossible to determine whether there was an intra-capsular fracture, an impacted fracture, or no fracture at all. But when I saw her the condition of things had altered, and the diagnosis was not very difficult. Upon asking her to bend the knee, as in any case of this sort you should do, because it affords a very good indication of what the damage is, I found she could flex the knee well and firmly, although with some pain. The muscles contracted in the ordinary way, and she flexed her knee up to a right angle easily and without great pain. If the case had been one of intra-capsular fracture, first of all she would not have been able to flex the knee to a right angle at all, and any attempt at doing so would have been very painful, mainly in consequence of the muscular spasm produced by the attempt at movement. This easy movement, combined with the slight eversion only, eliminates ordinary intra-capsular fracture, and the question now arises whether the case is a bruised hip or an impacted extra-capsular fracture of the neck of the thigh bone. If it happens to be an *impacted intra-capsular fracture* it is a very rare case, but the treatment of the two conditions of impaction makes it comparatively immaterial which it is.

A further point in favour of an extra-capsular impacted fracture is the large amount of swelling about the outer side of the hip, where there is a good deal of bruising. Taking all these things into consideration, it is pretty certain the case is not an intra-capsular fracture of the neck of the thigh-bone. There is a slight amount of shortening, and the question is, how much that shortening is? It is, in fact, only about half an inch. It is with regard to the shortening that I wish to speak to you particularly, because in the diagnosis of cases of injury to the hip the presence or absence of shortening is a very important matter. In this case, as you see, measurement by the tape at first shows no shortening, as the patient is lying now. But nevertheless there is, as I have said, some shortening. Let us, therefore, consider for a moment the fallacies which may arise in determining whether there is or is not shortening in a case like this. The measurement of the two sides is, you see, practically the same. But notice the difference in the two limbs. As I stretch the tape from the internal malleolus to the anterior superior spine, it rides on the injured side over quite a mound, which is produced by the blood tumour, whereas on the sound side the tape passes straight from point to point. The increase in length, represented by the raising of the tape by the blood mound, would negative shortening of half to three quarters of an inch, I suppose. It does not, therefore, follow in this case, because the tape stretched between the two extreme points on each side shows no shortening, that none exists. As a matter of fact, there is shortening, because if there were not the injured limb measurement would appear to be longer than the other, in consequence of the tape passing over this tumour. It is most important, in taking measurements to detect possible shortening in limbs, to be very wide-awake to the existence of possible fallacies, such, for example, as that just referred to. This, I believe, is an impacted extra-capsular fracture of the neck of the thigh-bone, and not an intra-capsular fracture. Such being the diagnosis, ought we to attempt to disimpact the parts? Ought we to pull the fragments apart, so as to correct, if possible, this small amount of shortening, or should the condition of the bone be left as it is? In impacted extra-capsular fracture, if the impaction is not reduced, there are two things, which mainly cause trouble. The first is eversion of the foot, since a patient who is compelled to walk with an everted foot is liable to catch the toe in all sorts of things, and may come to grief on that account at any moment. The second cause of trouble is the shortening; but this is so slight, as a rule, that it is hardly worth considering. The main cause for reducing the impaction is in

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order to correct the eversion of the leg. The question then arises, is it worth while in a case like this, where the shortening is so slight and the eversion so insignificant, to reduce the impaction? In point of fact, although it may be held by some to be very bad teaching, you will, I believe, be wise in a case like this, where the eversion is so little, to leave the fracture as it is; because you must remember that after the reduction of the impaction you have to treat the fracture *de novo*, and it is not at all certain that if you reduce the impaction you will bring about a result with less shortening than there is now, and you may, on the other hand, get other troubles due to your interference with a bone which is so surrounded with extravasated blood as this is. If the case is left alone the patient will be getting about comfortably as soon as the extravasated blood has disappeared, that is, probably in a fortnight, or at the most three weeks, an important matter with a patient who has to earn her living. This patient, if left alone, will have a good and useful limb; it will be a little short, and the foot will be slightly everted, but she will be able to get about quite well with a hardly appreciable amount of lameness. Whether in hospital work or in private practice, before you set about reducing such an impaction, place the whole matter candidly before the patient, and if he or she prefers to run a little risk in the hope of getting an ideal limb, you may be justified in reducing the impaction. But if the matter is left entirely to your judgment, do not be too eager to advise disimpaction unless the eversion is extreme; I have seen results follow disimpaction far less satisfactory than those which sometimes follow leaving the case alone. I do not wish to deter you in any attempt to obtain ideal results; but before rushing at the ideal, consider the matter a little from the utilitarian point of view.

Professor Gilbert (*Journal de Médecine interne*, August 1st) gives the formula for a method of administering zinc oxide in such nervous troubles as hysteria, insomnia, etc.

℞ Extract of hyoscyamus seeds. } of each 0·50
Extract of valerian. . . } gramme
Zinc oxide . . . . } (7½ grains)

M. Divide into 10 pills. From one to two may be taken daily.—*N. Y. Med. Journ.*

WITH DR. E. W. AINLEY WALKER AT GUY'S HOSPITAL.

General Pathology of Inflammation.

The process of inflammation constitutes, as we saw last lecture, the reaction to injury, and is the succession of changes which occur in living tissue when it is injured, provided that the injury is not of such a degree as to at once destroy its structure and vitality.

We considered the distinction between this process and the process of repair which is usually associated with its later stages, and saw that the former is present throughout the animal series, from the very lowest forms which have a differentiation of cell-groups, as a reaction of the mesodermal phagocytic group, and is exhibited by a similar series of events even in the simple unicellular amœba. On the other hand, the reaction of repair is similarly present in all animals, and is, indeed, more marked and more extensive in the lower series. It is a process in which all the cells, whether of epiblastic, of hypoblastic, or of mesoblastic origin alike take part, though in the higher animals it is usually very incomplete, and often represented chiefly by connective-tissue growth.

Accordingly we came to the conclusion that these two processes are quite distinct, and that repair is not included in the inflammatory reaction as at present understood, being only an associated and non-essential process; but we suggested that it might eventually prove that both these processes may be the exhibition of the same reaction to a stimulation of different cell-systems of the body.

On the question of the immediate cause of inflammation we have made reference to the conclusion of Hunter and his followers that it was brought about by an increase in size and number of the vessels of the part affected; to the epoch-making doctrine of Virchow that, like all other pathological processes, it was the expression of a succession of changes in the tissue-cells; and to the theory of Cohnheim that it is brought about by certain molecular changes in the vessel-walls.

We discussed the nature and causation of the vascular changes which occur in inflammation, and of the heat, redness, and swelling which are associated with these changes; and finally

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brought forward evidence to show that Cohnheim's theory of its essential nature was fallacious, by a comparison of observations on the inflammatory process in non-vascular areas with those in areas supplied with blood-vessels, and from the results obtained experimentally when fluids other than blood were circulated through the vascular system.

From the evidence which we thus obtained it follows that, though inflammation is not due, as Cohnheim thought, to alterations in the walls of vessels, yet such alterations form an important factor in the inflammations which occur in vascular areas.

Cohnheim believed that he had proved the dependence of inflammation on these alterations by the following experiment:—He produced injuries in the vessels of a part by circulating through them irritating fluids. On subsequently restoring the blood-flow through the part an inflammation of the neighbouring tissues followed. He concluded, therefore, that the injury to the vessel-walls produced the inflammation. The fallacy in this conclusion lies in the fact that there is no evidence nor any probability that the solutions which he circulated remained confined within the vessels and did not pass beyond the vessel-walls, injuring the neighbouring tissues also.

A simple stoppage of the circulation combined with the application of an agency normally quite innocuous may give rise to inflammation. This was proved by Cohnheim in the following manner:—The circulation in a dog's leg was arrested for half an hour by the application of an Esmarch's bandage, and the foot subsequently placed in water at 50° C. (122° F.), and the bandage removed. An acute inflammation supervened within some twenty-four hours.

This is to be explained as due not simply to an alteration in the vessel-walls, as Cohnheim thought, but to the fact that the vitality of all the cells of the part was so reduced by the lowered temperature, the deprivation of nutriment and oxygen, and the accumulation of carbonic acid and other metabolic products, resulting from the stoppage of the circulation, that a temperature of 50° C., usually quite harmless, became sufficient to produce a tissue injury and determine the development of inflammation.

Behaviour of the Corpuscles.—We must now examine the behaviour of the blood-corpuscles in the course of inflammation. As the circulation in the dilated vessels slows, the white corpuscles seek the sides of the vessels until the small veins become lined, as it were, or pavemented with leucocytes. Later, the red corpuscles also are affected. This change is brought about in part by an alteration in the corpuscles themselves or in the fluid plasma, and not entirely, as is often stated, by the increased adhesiveness of the vessel walls, for the former tend to adhere not only to the sides of the vessels, but to each other also. But the importance of the vessel endothelium in the process is shown by the experiment of Ryneck already mentioned, in which stasis could be produced when an indifferent fluid, like milk, was being circulated, but could not be induced at all after the vessel endothelium had been killed by circulating for a time solutions of metallic poisons, such as the sublimate of mercury.

As stasis approaches, the red corpuscles tend to run into rouleaux in the capillaries. This formation of rouleaux is a familiar event in blood outside the body in the presence of dead or inanimate matter, and is probably induced within the vessels in inflammation by the injured condition of the endothelial cells. Ultimately the condition of stasis becomes complete.

This may be followed by thrombosis, but only when the capillary walls have died does that event occur. Thrombosis is an intra-vascular coagulation of the blood, and is initiated, as we know, by a deposition of the blood-platelets originally described by Osler. In inflammation there is always a tendency to thrombosis. Thus if a frog's mesentery be exposed and painted lightly with a $\frac{1}{2}$ per cent. solution of silver nitrate, thrombi of leucocytes are formed in the capillaries.

The gradual slowing of the circulation leads to great engorgement of the vessels with corpuscles which are forced in by the arterial pressure, and shortly before the stasis is complete they may be seen to oscillate synchronously with the pulse-wave, moving a little forward with each beat and then recoiling in the intervals. But neither the slowing nor the stoppage of the circulation is the cause of the increased adhesiveness of the corpuscles. This is well shown in an experiment of Lister's which, however, had previously been used by Weber. He ligatured the vessel's of a frog's leg, thus producing complete stoppage of the circulation. It was now

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seen that the corpuscles in the vessels of the web moved freely among one another, and exhibited no tendency to adhere together. But when a tiny piece of mustard was applied to a part of the web free movement ceased in the affected area, and the corpuscles became adherent to each other and to the vessel walls. The result was an accumulation of corpuscles in the area, since any corpuscle which happened to glide in became adherent. If any corpuscle near the margin of the area chanced to escape from the zone of inflammation, it ceased to be adhesive, and again moved freely.

Stasis may be followed by complete restoration of the circulation; but if the vessel walls succumb and a thrombosis occurs a complete resolution is improbable. The thrombosed area becomes *ipso facto* extra-vascular, and necrosis, with the formation of an abscess, is the usual termination. The changes are very well exemplified in the formation of an ordinary boil. Here we can see a fiery red areola of increased circulation, with dilated vessels and a rapid blood-flow, from which the colour can be easily pressed out, but returns again immediately upon releasing pressure; within this ring there is a region of dull red or purplish congestion, in which the colour fades only slowly on the application of pressure, and returns slowly on releasing the compression—this is the region of dilated vessels with diminished rate of flow; and in the centre there may be a purple area from which the colour cannot be discharged by pressure—here is a condition of complete stagnation of the circulation, usually proceeding to thrombosis and necrosis of the central core of tissue. Even inflammatory thrombosis may, however, resolve completely.

Diapedesis.—Besides the intra-vascular events affecting the corpuscles, there is another phenomenon which they exhibit in the course of inflammation, namely, their passage through the vessel walls—a process usually termed diapedesis. This is exhibited first by the leucocytes, and subsequently by the red corpuscles also. The phenomenon was first observed in 1843 by Addison (of Guy's), and independently by Waller in 1846, though neither of them seems to have recognised its meaning. It was first studied by Cohnheim in 1867.

Shortly after the commencement of the "pave menting" of the smaller veins with leucocytes, numbers of them may be seen to pass through the vessel walls and come to lie outside the vessel, giving rise to the appearance known as "margination," and a similar process occurs in the capillaries also.

This passage of the leucocytes was thought by Cohnheim to be a passive process, for he found that stoppage of the circulation in the part by pressure on the main artery arrested its occurrence. This observation confirmed him in his view that inflammation was due to alterations in the vessel walls, since he concluded that the leucocytes which left the vessels had simply been forced out through or between the altered cells by the blood-pressure. The observation was, however, faulty, since, as we have already seen, diapedesis can and does occur after complete arrest of the circulation; and Metchnikoff observed it in the tadpole's tail after the heart-beat had been stopped by the action of curare, and the capillary circulation had entirely ceased.

The passage of the leucocytes is therefore an active process. They can be seen moving backwards and forwards from place to place along the vessel-wall before passing through, and when outside the vessel also exhibit active amœboid movement. Whether they pass out between or through the endothelial cells is not quite certain, though it seems more probable that they make their way through the intercellular material. There is no evidence that the leucocyte leaves any breach of continuity in the wall after its passage. It probably escapes much in the way that one soap bubble may pass through another without injury to either.

The passage of red corpuscles only begins after the commencement of the leucocytic emigration. There is no evidence that it is other than a purely passive process. The red cells are simply forced out through the vessel-walls by intra-vascular pressure, exactly as occurs in passive hyperæmia, and escape between the endothelial cells, frequently at a point where a leucocyte has previously passed out, and often several at the same point in quick succession.

Exudation.—Besides the passage of corpuscles from the vessels, there is in inflammation an increased escape of plasma, which accumulates in the tissue spaces of the area very much faster than it can be carried off by the lymphatics, and thus gives rise to what is called the inflammatory

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exudate. This is dependent chiefly, but not entirely, on the changes in the circulation. Not entirely so, because an active hyperæmia of non-inflammatory origin never gives rise to lymph formation greater than can be accommodated and carried off by the lymphatics of the part, and passive hyperæmia which gives rise to an obstructive dropsy does so by the production of an exudation of lower specific gravity than normal, while that of inflammation shows a great increase in solids. There is therefore another factor in the process besides the alteration in the circulation. This is the altered condition of the endothelium, which in its injured state appears to allow the passage of a more copious and more concentrated lymph than normal.

Virchow, who regarded inflammation as a process consisting essentially in an increased activity of the tissue cells in the surrounding region, due to the stimulation of the injury, attributed all the inflammatory changes to an increase of local metabolism. Both the alteration in the circulation and the exudation were in his opinion due to this increased metabolism. But such a view is quite untenable on various grounds. For increase of metabolism and accelerated tissue change could not in any way produce the essential slowing and stagnation of the circulation which occurs, and exudation is most marked precisely in those cases where increased metabolism is least in evidence, namely, at the beginning of inflammation, before any signs of growth and proliferation of the tissue-cells are present, and in those forms of inflammation especially where rapid and extensive cell destruction is most marked. Beyond the fact that we have in inflammation two factors—increase of circulation and an injured endothelium—both of which favour the escape of plasma from the vessels, we cannot at present go.

In certain modern text-books you may find it stated that the exudation subserves the purpose of diluting the irritant, of flushing out the inflamed area, and so forth. I must warn you against all such teleological explanations of the events which occur within the living body. No process which takes place in protoplasm does so in order to subserve a purpose. You might just as fitly say that water flows down from hill to valley to subserve the purpose of making rivers, which in turn subserve the purpose of diluting the sewage and flushing out the drains of towns along their course. All this is of course no explanation at all. It is worse than none, for it is most misleading, and may leave behind it an impression that something of scientific value has been said.

The processes which occur in living protoplasm, whether physiological or pathological, are, like all other events in nature, the effects of certain causes, and eventually lead to certain inevitable results, but there is nothing purposive about them. Processes which are unfavourable in their results tend to the destruction or deterioration of the organism, and so to its elimination in the course of evolution. Similarly processes whose results are favourable tend to be perpetuated in the race.

We cannot speak of purpose in regard to the exudation; it is a question of effect, not of intention; and absolute precision of language and of thought in dealing with these questions is essential to any scientific and accurate conception of the phenomena concerned.

Though it does not subserve the purpose, the inflammatory exudate has the effect of diluting the irritant if that irritant be chemical in nature, and of flushing out the area affected.

Of the causation of the exudate we cannot with our present knowledge go beyond the factors I have mentioned. We require first to understand the mechanism of ordinary lymph formation. This is a question which we cannot take up here, though on some future occasion I hope to deal with it in connection with the general pathology of dropsy; but I may point out to you that it is still uncertain whether the view of Heidenhain and his followers is correct which regards lymph as a secretory product, or that so ably defended by the researches of Dr. Starling, and which considers it to be a simple filtrate. In any case, however, we must remember that even if the process of lymph formation be one of mere filtration rather than of secretion, it is carried on through the substance of an animal membrane lined with living active endothelial cells, and must in any case be rather of the nature of dialysis than of percolation.

The lymph of inflammation differs from normal lymph not only in its greater volume, but also very remarkably in its percentage of solids. This is so much increased that the specific gravity may be almost trebled. Thus Halliburton, in some observations carried out upon the exudations in acute

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pleurisy and in hydrothorax, found the percentage of proteids in the former about three times that in the latter cases.

Inflammatory lymph also contains more corpuscles than normal, and may even contain so many red corpuscles as to acquire a faintly reddish tinge as it leaves the part affected.

The increased flow of lymph can be well observed in the hind leg of the dog, in which all the lymphatics of the foot unite into the superficial lymphatic trunks of the leg. And Cohnheim found by placing a cannula in one of these lymphatic trunks that when the foot was inflamed by the method which I mentioned in the first lecture, the flow of lymph was eight to ten times greater than on the healthy side. And this occurs without any venous obstruction or other passive congestion, which is entirely absent from this stage of inflammation, although the flow of blood is doubled. The only other condition in which a comparable volume of lymph is formed is in obstructive dropsy due to obstruction or pressure on a main vein, but in that case the percentage of solids in the lymph which leaves the part is very much diminished.

This experiment of Cohnheim's was repeated and confirmed by Lassar, who found the flow of lymph increased to eight times the normal quantity. Seeing, then, that the amount of proteids per hundred parts of lymph is almost trebled, it follows that the lymphatic circulation is carrying off from twenty to thirty times the usual quantity of proteid material.

Unfortunately it is not possible from these facts to determine very much about the composition of the exudate as it leaves the blood-vessels of the affected area, for besides the fact that the lymph which leaves the part contains an enormous number of leucocytes above the normal, which greatly alter its percentage composition in a chemical analysis, its proteids have also been increased by the addition to it of all the substances resulting from tissue degeneration in the inflammatory area.

Osmosis.—There is another factor which I must refer to which must play a part in the production of the exudation. I mean the variations in osmotic pressure. You know that if solutions of two substances or mixtures of substances which have different osmotic pressures be separated from one another only by an animal membrane a process of dialysis occurs tending to equalise the osmotic pressure on the two sides. Now the events occurring in the tissues of an inflammatory area must of necessity produce considerable and continuous alterations of the osmotic pressure in the tissue-spaces. Such alterations are probably of great importance in the causation of the exudate, although our knowledge is not as yet sufficiently advanced in this direction to permit of any very definite statement being made upon the question.

Inflammatory exudates differ in character and in appearance with the differences in the amount of *fibrin* formed in them. This varies both with the nature of the irritant and with the situation of the inflammation, and results in the production of the exudates described as serous, fibrinous, or sero-fibrinous according to their varying character. As regards the nature of the irritant itself, the result depends in part upon whether it is capable or not of causing extensive destruction of leucocytes; for, as we know from the physiology of the process of coagulation, fibrin formation is determined by the breaking down of leucocytes. And this we shall see depends very largely upon whether the inflammation is of the simple variety or is infective. The fibrin factors are always present in inflammatory lymph, though in amount which varies with the intensity of the inflammatory process; the amount of ferment present is probably determined by the extent to which the leucocytes undergo dissolution in the exudate. Hence, other things being equal, the amount of fibrin present depends on the extent of leucocytic destruction. It depends also on the extent to which the irritant at work possesses peptonising or digestive action on the fibrin formed.

As regards its situation, the character of the exudate is determined chiefly by mechanical factors. In dense tissue, as for instance, in the substance of a tendon, an extensive exudate is quite impossible, while in the loose synovial membrane which surrounds it everything favours the outpouring of a copious exudation in the tendon-sheath. Similarly in a loose areolar tissue exudation is able to proceed almost without restraint until the bulk of the fluid exuded becomes great enough to raise the extra-vascular tension to an equality with the intra-vascular, or even to exceed the latter, and thus produce compression of the smaller vessels and especially the veins.

In inflammation occurring in serous membranes

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the exudation is favoured both by the considerable vascularity of the part, and by the fact of the existence of a cavity with compressible contents which readily accommodates the fluid poured out. This is well seen in serous pleurisy, whereas in pleurisy associated with massive pneumonia, in which the lung becomes enlarged, solid, and incompressible, a great effusion is almost impossible.

The deposition of fibrin on an inflamed serous surface is comparable to the occurrence of thrombosis in inflamed capillaries, and can only occur after the degeneration or death of a proportion of the endothelial cells lining the surface of the serous membrane. Where a dense layer of fibrin lines the surface, this of itself mechanically tends to check the exudation from the vessels of the membrane.

The exudate when poured out is much less readily absorbed from serous cavities than from the ordinary tissue-spaces. This follows from the law of the relation of the volume of a cavity to its surface, since as the diameter increases the volume of the cavity increases as the cube of the diameter, but the area of the absorbing surface only as the square of that diameter. This helps us to an explanation of the tendency to slow absorption often exhibited by serous exudates, as well as of the accelerated disappearance of the fluid after a portion has been artificially removed, bringing the surface area once more into a reasonable relation to the volume of the fluid contained within the cavity.

Suppuration.—If the number of leucocytes normally present in an inflammatory exudate become so much increased as to convert the clear or merely blood-stained watery exudate into a thick and slightly viscid opaque fluid, we are accustomed to speak of the condition as one of suppuration, and of the fluid as pus. This condition, when arising naturally, is always due to the presence of bacteria, though artificially it may be brought about by certain other means.

Purulent fluid is usually yellowish-white in colour, but may be altered in appearance by the presence of blood-pigment or of blood-decomposition products. It may be stained by other colouring matters, such as bile-derivatives, or may obtain a special colour from the products of chromogenetic micro-organisms which it contains. Such is the blue or greenish pus produced by the action of the *Baccillus pyocyaneus*. It is alkaline in its reaction, it possesses a faint odour, and has a specific gravity of about 1030 (taking water as 1000), and it contains from 10 to 15 per cent. of solid matter, two thirds of which is proteid in nature.

The special characters of suppurative inflammations we shall discuss next lecture.

Gas Leakage.—According to Bayles, there is about 225,000 cubic feet of gas leakage to the mile per annum for the average 6-inch main, and in many cases a great deal more. It is also very explosive. He classifies the dangers from gas leakage into (1) fire and explosions; (2) asphyxiation; (3) blood-poisoning and anæmia. It is the latter class especially that is the most dangerous, and is practically the only ground for the domestic superstition about sewer gas. He thinks the enormous leakage of illuminating gas is accountable for the general prevalence of anæmic conditions in our large cities. The question is whether it can be stopped. We cannot depend on the companies, for leakage costs less than the means to prevent it, and nothing will really bring about its prevention except legislation.

Gonorrhœal Hæmorrhoids.—This term is used by Monroe, who thinks it might perhaps be better described as gonorrhœa of the rectum. The external appearance is often very similar to external hæmorrhoids; but if we cut into lumps we find no coagulations of the blood. They are frequently as painful as ordinary piles. There is a constant exudation of watery mucus, keeping the parts moist, and this helps in the diagnosis. There is much pruritus and burning, especially after defecating; the bowels are generally constipated. He reports a case and advises treatment. He would use calomel in one-eighth grain dose every four hours; also a saturated solution of boric acid, one ounce of this to the pint of warm water, injecting it into the rectum. When this passes out he injects nargol, ten grains to the ounce, requiring the last to be retained. This should be used twice a day. Twice a day he bathes the affected parts with peroxide of hydrogen, afterwards with liquid extract of hydrastis canadensis and a special ointment of calomel, ichthyol, one drachm of each to one ounce lanoline. For the tonic treatment he would use syrup of hypophosphites and the bitter tonics.—*Journ. A. M. A.*, Sept. 6th, 1902.

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THE TREATMENT OF TUBERCULOSIS.

A Clinical Lecture delivered at the Hospital for Consumption and Diseases of the Chest, Brompton, June 18th, 1902.

By F. J. WETHERED, M.D., F.R.C.P.,

Assistant Physician to the Hospital.

GENTLEMEN,—In order to satisfactorily carry out the treatment of any disease, it is desirable that that treatment should be founded on certain principles, which in their turn should be thoroughly and systematically carried out. In days gone by we relied upon our treatment almost purely from an empirical point of view, but of course it is much more satisfactory if we can establish a rational mode of treatment. Some of you may have seen Dr. Mitchell Bruce's book on the 'Principles of Treatment,' and I think those principles which he there lays down are perhaps the most convenient, if not altogether the most scientific arguments which have yet been deduced. He bases all treatment on three principal grounds, namely, ætiological, pathological, and symptomatic.

As types of those diseases which may be treated on ætiological grounds we have those diseases which are caused by pathogenic organisms. A very good example of a disease which may be treated on pathological grounds is bronchitis. In that malady, in the early stages, we find swelling and dryness of the tubes, and accordingly we give the depressant expectorants, such as ipecacuanha or antimony. Then, in the second stage, when the mucus is free, we give more stimulating drugs, such as carbonate of ammonia, squill, and so on. If the secretion is too free, we give acids to diminish it. Next, with regard to treating disease on symptomatic grounds, we have numerous examples of this, such as tuberculosis, in which we have to treat the diarrhœa, the vomiting, and the night-sweats, and we have to relieve the cough.

When we come to the treatment of a case of

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tuberculosis we have all these elements very strongly before us. We have the ætiological principle founded upon the presence of the tubercle bacillus. We have also the pathological features, because we have to encourage fibrosis, we have to reduce the temperature, we have to encourage hypertrophy of the lungs, and to encourage the keeping up of repair of tissues. We have also the symptoms to treat—the cough, night-sweats, the diarrhœa, and so on. When we come to treatment based on ætiological grounds we have one thing which we must most particularly bear in mind. When Koch discovered the tubercle bacillus in 1882 the medical profession as a whole rushed to the conclusion that if we could destroy the bacillus in the lungs we could cure the disease. A great number of investigators at once set to work upon this. Unfortunately, however, we know that up to the present time, though it is twenty years since the tubercle bacillus was discovered, we have not found a sure means of directly destroying the bacillus in the lungs.

Some of you may have read Dr. Howship Dickinson's address at St. George's Hospital, in which, basing his remarks on the parable of the sower, he gave an eloquent discourse on the seed and the soil, showing that medical men are, and have been, bestowing more attention on the seed than on the soil, and that their treatment in that direction had failed. This soil varies in different people, and its condition is known by different terms. Some call it the "second ætiological factor," others call it diminished resistance. The tubercle bacillus alone will not cause tuberculosis. If that were the case, the human species would long ago have been extinct. We need for its spread the particular soil upon which it will grow. It is perfectly logical to try to destroy the bacillus, but it has not been found practicable, and, as I have said, all efforts in this direction have failed. On the ætiological ground, the first idea in modern times would be to seek for an antitoxin; to try to get either from cultures, or from human beings or animals affected with tuberculosis, an antitoxin, in the same way as the antidiphtheritic or antitetanic serum. Koch some years ago hoped that he had discovered this antitoxin in the shape of tuberculine. Unfortunately for him, however, his secrets were forced from him rather too soon, and, as we know, the whole thing turned out to be almost a fiasco. Perhaps medical men tend too much to cry down the effect of tuberculine. Even now we have some observers bringing forward their views upon this substance, and we have some authorities who maintain that in certain cases tuberculine has a very good action. One of the observers brought forward this point: that if we can examine the sputum in certain cases of tuberculosis, and we find in that sputum only the tubercle bacillus, then the effects of tuberculine are certainly favourable. But we know that destruction of the lung depends on the presence in the lungs of a large number of other organisms, such as streptococci and staphylococci, and that it is their action as well as that of the tubercle bacilli which produces this destruction of lung. It is obvious that in such instances tuberculine could have had no definite remedial effect. I must say that in my experience, and I think in the experience of most of the physicians of this hospital, it is uncommon in young subjects to find cases in which the tubercle bacillus only is present; but we find such a condition in elderly people. But what is the course of the disease in elderly people? We find that they have a comparatively low temperature, that the disease runs a very slow course, there is a tendency to fibrosis, that the damaged lung tends to heal itself; and it is in these very cases that tuberculine is said to be of value,—that is to say, in those cases which tend to heal naturally, while in those cases in which there are other micro-organisms present also there is far more destruction of lung.

This argument may be carried further. In trying to treat tuberculosis we have a disease which we know is absolutely curable not only as viewed from a clinical, but from a pathological point of view. I have taken out a number of statistics from the cases at the Middlesex Hospital, following upon that of my colleague Dr. Kingston Fowler and Dr. Sydney Martin, who was formerly attached to that hospital. Taking the post-mortem records for a large number of years at the Middlesex Hospital of people dying from diseases other than tubercular diseases, we find that no less than 9 per cent. of those cases have had pulmonary tuberculosis at some part of their lives, and it has become completely healed. We find various signs in the apices or in other parts of the lung. These signs may be in the shape of cavities which are obviously tubercular, some of them still caseous, although cut off from the rest of the lung by fibrous tissue. So that 9

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per cent. of these people get well spontaneously, and in a large number of such cases the patient becomes as well as if he had never had tuberculosis. We are therefore dealing with a disease which is perfectly curable.

Since the discovery of the bacillus by Koch, members of the medical profession have attempted in many ways to kill the bacillus after it has become engrafted on the lung. The obvious way in which medical men tried to do this was by means of the inhalation of various germicides. This has proved an utter failure. We know as a fact that there must be a very great difficulty for these germicides in vapour to reach the part of the lung in which the disease is situated. We know one of the earliest signs of pulmonary tuberculosis is weakness of breath-sounds at one apex, and that means that the lung at that apex is not expanding so well, and naturally air is not entering into it; and if that is so neither can germicide vapours enter it. So we are met with that difficulty at the outset of such treatment, that the germicides must have great difficulty in reaching the diseased portion of lung. Post mortem we see part of the lung is filled up by fibrous tissue, that a great number of bronchi are obstructed, and many of them destroyed. There, again, it is impossible that a quantity of air can reach the apices. Therefore there is no reason for surprise that attempts to cure pulmonary tuberculosis by the inhalation of germicides have proved a failure. Moreover germicides can only act when they are in certain doses, and in order to kill bacilli in the tissues of the lung the germicides would have to be given in considerable doses to produce this effect. Probably if these doses could be given they would do more damage to the host than to the bacilli themselves. For instance, creasote has been employed. Owing to its properties, it can be given in only small doses, and in nothing like the potency which would be necessary to kill the germs. Oil of peppermint, terebene, menthol, and even hydrocyanic acid in the form of inhalations have been tried, but so far without any marked success.

The next way in which germicides have been tried is by absorption into the blood from the stomach. Various preparations which have the power of destroying tubercle bacilli in the laboratory have been given by the stomach in the hope that they will be absorbed and taken by the blood into the diseased tissues. There, again, in order to have effect they must be absorbed in certain doses. Two questions come in in this connection: Is the dose which is sufficient to kill the bacilli likely to be harmful to the patient? and the second is, Can the drug be absorbed in sufficient quantity without decomposition to reach the affected portion in proper strength? I can only say I have myself tried many drugs, in both small doses and in large doses. I have tried creasote very extensively, guaiacol, carbonate of guaiacol, and various other products of the coal-tar variety. My experience is that those patients who take those drugs improve as a rule, at any rate for a time. But I may be rather heterodox in my views as to how they improve. I consider that those patients improve not because those drugs are absorbed and carried to the lungs, but because, having active germicidal properties, they destroy the germs of fermentation in the stomach and intestines, and so aid the proper absorption of the food stuffs. My experience has been that those patients who take three minims of creasote three time a day do as well as those who take heroic doses of twenty minims three times a day. My conclusions may be wrong, but there is no doubt that patients who have small doses of this substance certainly do improve.

Another way which has been tried is by injection of substances into the veins. Many of those experiments are still *sub judice;* we have come to no conclusion about them. We have this great difficulty, that we do not know exactly what becomes of the substances when they get into the blood at the body temperature. Perhaps the patient has had a very high temperature, and consequently changes may occur before the drugs reach the lung. Some of them cannot exist at a temperature of 103° or 104°, so we cannot sterilise them by heat before we give them. Perhaps at such temperatures they may be perfectly inert substances as regards action upon the bacilli. It must be confessed, then, that hitherto the use of drugs for directly killing the bacilli has only been attended with failure. It is rather a depressing confession to make, that although during these twenty years such numerous experiments have been made we are very little further as regards directly killing the bacilli in the lung than we were before.

But there are other methods of treatment which

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have been attended with greater success, and which we hope to see in the future produce even greater success; that is to say, not trying to absolutely kill the enemy, but to make the country in which the enemy has become established completely unfit for his occupation—to try to place the tissues in such a condition that the bacilli will not flourish upon them, and so that consequently they die of starvation. We must have certain conditions of the tissues for the bacilli to flourish, and immediately we can put the tissues in that condition so that the bacilli will not grow we have done a great deal towards curing consumption and preventing it. But to do this the patient must be placed in hygienic surroundings in every way, and we must particularly see that his digestive and absorptive processes are going on properly. There is a certain medicine in our hospital pharmacopœia which for some reason is about the only mixture which has stood the test of time. It is the "Mist. Gent. Alk.," consisting of three minims hydrocyanic acid, fifteen grains of carbonate of soda, and aromatic infusion of gentian. That has been given almost ever since the hospital has been established. Why is that so successful? I believe it is because it places the stomach in such a condition as to readily digest the food in an efficient manner, so that it will be absorbed. That perhaps was the earliest start of the hygienic treatment of tuberculosis, now better known as the "open-air" treatment of tuberculosis, or what some observers call the "sanatorium" treatment of the disease. And it is more particularly upon that subject that I wish to speak to you to-day, and I hope that my remarks may not be already known to you in their entirety. I shall speak of the treatment not only in sanatoria, but in general practice, because there is no reason why patients should not be treated as well in private practice by open-air or hygienic methods as they are in big sanatoria. What are our objects in this hygienic treatment? We have three main objects in view. The first is to stay the activity of the disease by increasing the resistance of the patient; the second is to promote and encourage the reparative process, that is to say, to increase the growth of fibrous tissue; the third is to maintain the heart power and develop compensatory growth of the damaged portions of the lungs. Those are the three main principles upon which this treatment is based. What, then, are the methods for carrying out those principles? There are several, which I will just mention, and then we will consider them seriatim.

The first requisite is the residence of the patient under medical supervision. This "under medical supervision" I would lay stress upon. Some practitioners seem to think that if you place patients under certain conditions and only see them once a fortnight, that is sufficient. It may be sufficient in the later stages of the treatment, but certainly it is not enough in the earlier stages. It is absolutely necessary in the first few weeks of treatment that the patient should be seen at least once a day, preferably twice or three times a day. Hence the advantage of sanatorium treatment, where the patients are seen by the medical officers very frequently. Here I may add that this sanatorium treatment has two advantages: not only has it the advantage of curing the patient in many instances, but it is also educational. It shows the patient how he should live, and the manner in which he should conduct his life after leaving the sanatorium. But there is no reason at all why the same instructions should not be given to him by his medical attendant. Still, in the early stages at any rate, the patient must be seen frequently by his medical attendant, and the reasons for that I shall state presently. The second point is complete freedom from excitement of all kinds. Every excitement must be avoided; the patient must live as quiet a life as possible. There again the sanatorium has advantages over the private treatment, for the patient is removed from his friends, and placed in such surroundings that he can remain quiet. Thirdly, the life must be spent entirely in the open air, as far as the exigencies of our climate permit. Next, the patient should have rest during periods of the disease attended by fever. Fifthly, he should have methodical exercises, especially hill-climbing. Sixthly, there should be an abundant varied dietary. Seventhly, there are various hydrotherapeutic measures; and lastly, life under these circumstances should be rendered as pleasant as possible, and that is a provision which often is not laid enough stress on. Those of you who have had experience of treatment know that rendering life as bearable as possible is by no means a slight part of the treatment, and it is on that point very often that the treatment may break down.

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Perhaps the easiest way to go through these points is to trace out how the patient should be treated when he comes under our care. First of all a word or two with regard to those cases which are likely to benefit most. Those patients with large cavities can hardly hope to attain any very great relief. The relief which can be produced under those circumstances must be extremely modified. Those patients who do best are naturally those who have only one apex affected, and who have a moderate degree of fever. A fair number of cases, even with both lungs affected, do well, and even some of those cases which have advanced to the cavitation stage. Those cases where there is simple consolidation do best. When a patient comes under treatment the first thing should be to put him to bed, and keep him there for two or three days, his temperature being taken two or three times a day. It has been found that a more exact temperature can be taken in the rectum than in the mouth. The axillary temperature is practically never used in hospitals for chest diseases, and those who have had large experience in these matters state definitely that you can judge more certainly by means of the rectal temperature than by any other. Having observed the patient two or three days in bed, we know the range of his temperature, and it is in this observation of the temperature that medical supervision is so important. The patient's life is practically governed by his range of temperature. During these first two or three days we also commence to diet him. A very great change has come over medical men with regard to diet and temperature in tuberculosis. A few years ago if a patient had a temperature of 103° to 104°, he was placed on liquid diet; but in the open-air treatment we do not regard temperature when we come to diet. The patient is given the same diet when his temperature is raised as when it is normal. Therefore, when we observe a patient in bed we should at the same time begin to instruct him as to what articles of diet he is expected to eat. Even in those two or three days we begin to push the food. The patient should have three good meals a day, and I think it is best to give nothing in between. Let him have for breakfast bacon, eggs, fish, ham, or tongue, and so on, with coffee, and at the same time at that meal he should drink about a pint of milk. For lunch he should be graduated up, so that he has equal to a leg and wing of a chicken, two large mutton chops, or three big slices off a joint, with a full amount of vegetables. After that he probably has some wholesome pudding, such as bread-and-butter pudding or milk pudding. He should have two tumblers of milk and two ounces of cream. For the sake of companionship he may have in the afternoon tea, but that is not included in the regular programme. For dinner he should have much the same diet as for lunch, though perhaps it should not be quite so heavy a meal. A strong point is that he should have a good meal in the middle of the day, which should embrace freshly cooked meat. That meat I look upon as rather important, because various errors of diet may arise in connection with re-cooked meat. We have to lead up to these meals very much in the same way as under the Weir-Mitchell treatment.

Now comes the difficult part of our treatment—the taking of exercise by the patient. As long as the patient's temperature rises in the twenty-four hours to above 100° he should be kept in bed entirely, and should not take any exercise at all. As soon as his temperature ceases to rise to 100° in the twenty-four hours he may rise from bed and lie upon a couch in the room, and if possible his couch may be pushed on to the verandah. As soon as his temperature falls below 99·4° his real education in these matters commences. He is told the first day to dress himself and walk to a seat about fifty yards from the house, to remain there half an hour, and then walk back again, the rate of progression being about two miles an hour. As soon as he returns his rectal temperature is taken. If the effort raises his temperature above 99°, he must not take any exercise on the next day. If that fifty yards' exercise does not raise his temperature at all, he is next day allowed to take a hundred yards' walk. And so his exercise is gradually increased, the temperature being all the time carefully watched. This exercise is allowed to go on until the patient can take an astounding amount, some of them do twelve or more miles a day. The medical man at the early stages must be prepared to give such instructions. A prescription must be given to the patient every morning, but instead of a prescription in bad Latin it is given in English, and consists of instructions where he is to walk and how long he

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is to be out. The walking is to be at first on level ground, and subsequently he is told to walk up a hill. This walking up-hill develops the heart power, and it also develops certain portions of the lungs. Hill-climbing is considered an indispensable part of the treatment, and so for the patient to undergo the hygienic treatment properly he must live in a slightly hilly country. Of course, when this routine has been undertaken for two or three months regularly, the patient gets to know himself by his range of temperature how much exercise he should take. A careful watch is kept as to what effect the exercise has upon the temperature, the patient being made to rest either entirely or partially, according to the height of the temperature. After he has been under that restricted treatment for some time he is allowed to take more freedom. He may have friends to visit him, and he is permitted to take part in certain recreations. And there comes the great difficulty—the recreation of the patient. One finds again and again when a patient is in a sanatorium that he insists upon leaving, and says he can stand it no longer, but he will do his best to carry out the required treatment outside the sanatorium. He probably goes away and indulges in too much exercise, and the result is he collapses. I think medical officers of sanatoria might do well to bear in mind the words,—

> "Sweet recreation barr'd, what doth ensue
> But moody and dull melancholy?"

Patients who are undergoing this treatment day by day want recreation; but it taxes one's ingenuity very much to know what form of recreation should be given them. The recreation must be such as to take them into the open-air, and for certain patients we want it to be one which shall include a certain amount of exercise. For these the amusement to be recommended *par excellence* is no doubt croquet under the modern rules. It takes a tremendous time to play a game—a four-handed game takes about four hours. In sanatoria, by organising tournaments, we can keep our patients out of doors and keep them interested for a proper length of time.

The time during which a patient should remain under treatment is four to six months; four months should be the minimum, and it should preferably be six months. In the case of private patients I strongly advise you to tax your ingenuity to amuse them, so as to avoid introspection, and to prevent them continually discussing their temperature charts. As the patient gets on in the treatment a little more exercise can be allowed, such as bicycling on the level, quoits, and moderate golf. The full swing of the club may be harmful, at any rate until the patient has got some way in the treatment. Our great trouble is to prevent the life becoming monotonous. Of course I am treating these matters sketchily, as time will not permit of my doing more.

Now with regard to the full access of air, we find, especially amongst ladies, that if we suggest that the windows should be kept open while the patient is asleep, and also during the day, she will say, "I am sure I shall catch cold." You can reassure her on that point, and ask her to let you know if she does. As a matter of fact these people rarely take cold. In the case of private patients you have the doors open and the windows a little open, gradually increasing the amount until eventually the patient sleeps with the windows wide open. The best kind of windows for these patients are the French casement windows. When there is wind and driving rain these can be protected by louvre shutters, and the supply of air into the room can be ample. What we want for these patients is a room which will ventilate itself by the window, and we want a through draught. In private houses it is a very good procedure to get some staple to fasten the door so that it can be fixed open, and so that there is consequently a constant draught passing through. In sanatoria the doors are constructed with a fan-light over them, each door being opposite a window in the passage, which enables us to get a constant current of air. There comes another striking fact, that the patient should take this exercise whatever the weather may be; wet weather itself does not signify. A tuberculous patient may go out in the pouring rain and not suffer for it, and in some of the German sanatoria the patients do not trouble to change their clothes. But a strong wind will at once cause a tuberculous patient the greatest inconvenience. Therefore, in giving our prescription to the patient we are very much guided by the wind prevailing at the time. We observe which way it is blowing, and we send our patient for a walk upon which we know he can be protected from the wind. In sanatoria grounds shields

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are put up, so that the strong wind may do the patients no harm. In this country the only thing, practically, which will give these patients cold is a strong north-easterly wind.

With regard to the hardening process which patients undergo at that time, it is very remarkable. I remember going to see a sanatorium in the middle of January. I had to drive seven miles, and the weather was such that I was very glad of a warm coat. I arrived before luncheon, and I was asked if I would like to lunch with the patients. I said yes, and they gave me a full patient's diet. After lunch I was taken for a walk to see the grounds. When I came back some patients were playing a croquet tournament, and others were looking on. I and my friend had our thick coats on and were very glad of them, but amongst all those patients there was not a hat or a great-coat. That shows how a constant life in the open air will harden patients.

That is very briefly a sketch of the plans to be adopted in so-called hygienic treatment, the main principles of which are life in the open air, protection from the winds, the enforcement of a very full diet, with plenty of fatty food and milk, and graduated exercises according to the temperature.

Glioma of the Nerve Centres.—Oberthür has been enabled to examine seven cases of this nature in the laboratory. Macroscopically, the appearance of glioma was sufficiently characteristic to differentiate it from all other tumours. It was always clearly translucent, except in the degenerated parts; the colour was rose or yellowish grey; its consistence often very diffluent, even gelatinous; it was never encephaloid. A great number of small vessels coursed through it, giving place at times to central hæmorrhagic suffusions. Although the tumour could never be enucleated, it was sometimes limited by a pseudo-wall of dense neuroglia. Outside of this wall the infiltrated gliosis was like that which surrounded all foreign bodies in the brain not having the aspect of a neoplasm. In one case which had existed for three years the only symptoms were headache with optic neuritis and amaurosis, permitting no localisation. Other cases had a more destructive course.—*Medical Record*, September 13th, 1902.

WITH
SIR LAUDER BRUNTON
AT ST. BARTHOLOMEW'S HOSPITAL.

GENTLEMEN,—We have had in the wards lately several cases of disease of the cerebrum, and it is to two of these especially that I propose to-day to direct your attention, because in those two cases we have had the advantage of having a post-mortem examination. There are two others in the wards which you might look at. One in Elizabeth Ward is a case of tumour, probably either in the cerebellum or somewhere about the crura cerebri. Another case is in Rahere Ward—a boy with what is probably tubercular meningitis.

We have had within the last week, also, two operations upon the head. One of those was in the case of a man who was operated upon about a year ago, but who returned to the hospital in consequence of the symptoms having come on again, and in whom the operation was repeated. This patient is a warehouseman, and began to suffer from pain in his head in February of last year. He was admitted to the hospital on May 13th, and he was then suffering from pain in the head and inability to walk straight. He had no staggering gait, but when he tried to walk straight he always bore to the right; so that if he started to walk down the middle of the ward, he found himself running into one of the beds on the right side before he had proceeded far. The pain in the head was at first comparatively slight, and when we proposed to him to have an operation he at first objected. But by-and-by the pain in the head became so excruciating that he became exceedingly anxious for the operation, and counted the hours until Mr. Walsham should be able to operate. This inequality in the gait seemed to point to something wrong in the cerebellum, but I was uncertain as to which side of the cerebellum was implicated, because a slight lesion has got a different effect from a severe one. An irritative lesion has an exactly opposite action from a paralysing one, and I was not quite certain what the nature of the lesion was. The pain was so intense, however, on the left side of the occiput that we decided to operate upon the left side, and Mr. Walsham accordingly trephined over the

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cerebellum, removing around the seat of the trephining a good deal of bone by means of pliers, and cut through the dura mater. The cerebellum then protruded very much into the wound, and I thought in all probability the lesion was a glioma. In gliomata you very often find that the interior of the tumour is converted into a cyst, with a considerable amount of tension. The tension may be so great that in one child under my care, which was operated upon a good many years ago, when a trocar was run into the cerebrum, the liquid spurted out to a distance of at least eight or ten feet. Mr. Walsham put the trocar into the cerebellum, but no liquid issued, nor could he find, on moving the trocar about, any hardness or resistance indicative of tumour. Some of the cerebellar tissue projected and was removed, and the wound was sewn up. The immediate effect of the operation was to relieve the intense pain from which the patient suffered. The wound healed well, and he was sent to Swanley to recuperate on August 3rd, that is about six weeks after the operation. At that time he appeared to be almost well; the pain had quite gone, and he was able to walk perfectly straight. After he had left Swanley, the pain came on again slightly, especially upon exertion. He was readmitted on October 8th, but, as we did not think it advisable at that time to operate again, he was discharged on November 24th. Since that time he has had almost continually occipital headache. During the last three weeks before readmission the headache became much more severe, and accordingly he was taken in again on May 5th. He was operated upon on Friday, June 1st, and Mr. Walsham removed a piece of bone from over the cerebellum on the right side in the same way as he had done before on the left side. He again inserted a cannula, and with the same result as on the left side, that is to say, no fluid came out, and he met with no resistance indicative of any tumour. So we are still in the dark as to the exact condition. I cannot help thinking that there is a glioma somewhere, and I fear very much that the prognosis is by no means favourable. I have very little doubt that the present operation will relieve the symptoms, as it did before, that it will lessen the pain, and make the patient feel comparatively well for a short time, but I fear that there will be a return of the pain, which very likely will be accompanied, when it next appears, with some interference with the mental processes, and by-and-by it will turn out that the patient is doomed. But still there is no doubt whatever that the correct thing to do was to repeat the operation which had given so much relief before.

The other patient who was operated upon was a school-boy æt. 10. He had been well until eighteen months ago, when he fell and bruised his nose. A month later he suffered from headache across the forehead, over the right eye, and round the right ear. He had also vomited. The headache continued and was getting worse. There was a vague history, in this case, of inflammation and discharge from the right ear, and he was apparently somewhat deaf in that ear. On examination of the ear, however, the tympanic membrane was found to be perfectly intact; it looked whiter and more opaque than usual, but was not bulging, and was otherwise perfectly natural. On pressing behind the mastoid bone he complained of a little tenderness. This also was somewhat uncertain. There was a good deal of optic neuritis, and he had already had the two other prominent symptoms of cerebral tumour, or what comes almost to the same thing, cerebral abscess; that is to say (1) severe headache, and (2) vomiting, and now there is associated with those two (3) optic neuritis. The optic neuritis was so severe that it interfered very seriously with vision, and was becoming daily worse. In order to save the vision, more than for any other reason, it was decided to operate upon him at once. I was a little uncertain as to where the operation had best be performed, whether it would be better to just cut over the mastoid, so as to open, if possible, into any of the cells, and drain if any pus could be found. But the main point here was really to relieve the tension within the brain. When Mr. Power operated behind the mastoid the brain was found to be projecting, and the patient has certainly been relieved by the operation, the tension having been lessened.

These are two occasions on which patients have been subjected to operation and in which the operation, though in neither case has it been curative, has certainly given relief.

The other cases, which are still in the wards, we will not take into consideration to-day.

Of the cases which died one was of considerable interest, inasmuch as it was very rare, and it was a

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case the nature of which, but for the post-mortem examination, I doubt if we should have suspected. This was the case of George S—, a footman, æt. 18. The history was that he was in good health as usual until Thursday, April 25th, when he complained of headache, he vomited, and was thought to have a bilious attack. On inquiring more carefully afterwards, we found that this was not quite correct. We found that he had not been in his usual health for some time. One does not know what he had complained of, but he had not felt well, and had been showing symptoms of considerable irritability for a good many weeks before. He seemed to be a healthy man, but when admitted he was distinctly dull and apathetic. He did not care to be moved, but lay still upon his side, and if we tried to move him at all he seemed to resent it. When admitted his temperature was 103·5°. He was seen by a doctor before admission, who thought that he might very likely be suffering from typhoid fever, because in that disease you get high temperature, headache, and apathy. Practically it would have been very hard to say whether it was typhoid or not. There was nothing to be found on examination of the abdomen, nothing in the right iliac fossa, neither gurgling, tenderness, nor swelling. There were no spots, but we could not have been quite sure of our diagnosis without trying the Widal reaction. This was tried, but the result was found to be negative. But even this does not show positively that a given case is not typhoid, because we have had in the wards one very well-marked instance of typhoid in which we tried the Widal reaction several times, always with negative results; yet, unfortunately, in that case there was no doubt about the diagnosis of typhoid fever being correct, because the patient died, and we found typhoid ulcerations in the bowel. The temperature in this footman's case was not quite like that of typhoid, because it ran a much straighter course. The typhoid temperature oscillates a little, but it does so within very narrow limits, not more than 1° to 1½°. Altogether it seemed very much more like a case of something cerebral than typhoid fever, especially when we found Widal's reaction was negative. But if it was a case of cerebral disease was it due to the action of toxins on the brain, or to some organic lesion of the brain? There was no indication of anything likely to lead to toxic poisoning in the condition of the kidneys, because the urine, although containing a cloud of albumen, had a specific gravity of 1028, and was in ordinary quantity, and free from sugar. It seemed, therefore, that the lesion was something of an organic nature, but what it was I could not say. There were no localising symptoms, so one could not dream of operating. Gradually the patient became a little worse, respiration became very rapid, the pulse was 104, the temperature still high, and the patient became more completely comatose. Then the respiration stopped, and there were the usual symptoms of death by sudden stoppage of respiration, the patient becoming blue, and the jaws becoming clenched. The pulse rate increased until it could not be counted, and then it stopped, and the patient died. On examination of the chest one or two bronchitic râles were heard, especially on the right side. We attached no importance to these at the time. On examining the thorax, practically the only thing that was found was at the bifurcation of the bronchi, and there, occupying the position of the usual lymphatic gland on the right side, was a rounded swelling containing thick purulent matter. It was about half the size of a golf-ball; the cavity of the abscess was thoroughly explored, and led to nowhere in particular. Here, then, was a focus of infection. How it had arisen we could not say. Another case has been described similar to this, where a large gland was found in the thorax, which had also led to a pyæmic infection, but in it the enlargement of the gland was consequent upon pneumonia, whereas no such cause was present in ours. On examining the brain in our case a thick layer of lymph was found at the base, but the most remarkable condition was in the brain itself, for on slicing the brain across it was found studded with numerous small abscesses, most of them about the size of a hazel nut, and filled with thick pus, having a slightly greenish tinge. These were very evident as they stood out against the white surface of the brain substance. The brain has been hardened, and the characteristic appearance has to a great extent disappeared, because the pus is now washed away, and all that is to be seen is simply the series of slight excavations in the brain substance where those abscesses were. These were very numerous; there were probably a couple of dozen of them. In the other case I mentioned there were only six. Probably

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the abscesses here were more numerous than in any other case which has been reported. The abscesses here were undoubtedly pyæmic, and on microscopic examination a large number of streptococci were found, almost a pure cultivation. I did not know exactly how this infection had come about, and I do not know now; but taking into view the other case recorded by Drs. Finlay and Adam, where there was also a large lymphatic gland at the bifurcation of the bronchi as the apparent source of the pyæmia, I think it is possible that this youth may have had influenza with some slight affection of his lungs. Somehow or another this gland in the thorax had become infected from the lungs, and had led to secondary pyæmia, the matter from the gland having got into the circulation and become distributed by the heart through the carotids into the brain, because both sides of the brain were equally affected. At first I thought it possible that the patient might have had gonorrhœa, with some abrasion in the urethra through which the gonococcus had been absorbed. But careful search was made for the gonococcus in the pus contained in the abscesses, and no such organism could be found. So that, failing any evidence of the gonococcus, I am inclined to suppose that he had had some slight pneumonia consequent upon influenza, and that this had formed a source of infection in the bronchial gland from which the brain had been affected.

The other case to which I wish to draw your attention is that of an old woman æt. 71, who was a monthly nurse. She was admitted on April 30th, with paralysis of the left arm and slighter paralysis of the left leg. The history was that on April 30th she began to make grimaces, and that on one occasion she fell off her chair. What happened within the next hour or so one does not know, but some people came into the room and found she was conscious and able to talk, but that her left arm and left leg were weak. She was brought into the hospital, and on examination it was found that she was conscious; she could speak and answer questions, but she was drowsy and disliked being disturbed. Here, then, we have a case of hemiplegia, but the question we had to decide, if possible, was whether the hemiplegia was due to atheroma and plugging of one of the cerebral arteries, or whether it was due to rupture of a cerebral artery and the discharge of blood into the brain substance,—in fact, apoplexy. When I first saw her I was inclined to say it was due to atheroma, and I thought it was due to thrombosis of one of the cerebral arteries rather than to rupture of an artery. My reason for thinking this was that the arm was affected more than the leg, and the affection was more or less of a temporary nature. It had partially improved from the time she came in. That was not likely to have occurred if one of the vessels of the brain had actually broken. But still, I was not quite certain of it, and when we came to look at the various factors which might have enabled us to decide, we found them very equally balanced in favour of atheroma on the one side and of apoplexy on the other. With regard to her age: in old people apoplexy is very frequent, but so is atheroma. With regard to family history: she had one sister who died of apoplexy at the age of fifty, and another who died of heart disease at the age of forty-nine. On examining the heart, we found a distinct systolic murmur over the base, but no diastolic murmur, and this seemed to point fairly distinctly to the existence of atheroma in the aorta. We found also that her heart was enlarged, and that there was a loud, mitral, systolic murmur at the apex conducted towards the back. Therefore, we had to deal with a hypertrophied heart and atheromatous arteries. So we had factors which would do for either of the diseases I have mentioned. The atheromatous arteries themselves might very well give rise to either embolism or thrombosis, so it was not very easy to decide. She gradually improved a little, but in a few days afterwards she became worse again. On May 2nd she began to complain of a good deal of headache, and her general condition was worse than on admission. On the 3rd she gradually became unconscious, and died on the 7th. I then altered my diagnosis, and thought that from the rapid death of the patient we had probably had to deal with rupture of a vessel rather than with its plugging. Upon post-mortem examination we found that this was not the case; what really had occurred was extension of the plug, and extensive softening From the specimen you will see the amount of softening which had occurred. There is softening at several spots, the plug being so large as to block the middle meningeal. Post-mortem also cleared up several other points about the case. The murmur which was heard at the

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base of the heart was due to the aorta having become exceedingly atheromatous; it was rough, and gave rise to an obstructive murmur. The valves were perfectly normal and closed perfectly, so that there was no backward regurgitation. The mitral orifice was surrounded by a rigid calcareous ring, but the valves were not much shrunken or thickened. The thoracic aorta was very much affected by atheroma, and from it plates had apparently been detached, which had affected the kidneys to a very great extent. The left renal artery was calcareous, and one of its branches was thrombosed. Infarcted areas were found on the surface of the kidney, which had a marked appearance on section owing to extensive necrotic areas. The right renal artery was calcareous but contained no thrombus. From this condition of the kidney the urine was albuminous. You will thus see the power that atheroma has to affect different parts of the body. Here we had both the kidneys and the head very much affected by atheroma. It is a condition which leads to a great many affections which are often unsuspected, but it is one which you should keep your eyes open for, and especially during the hot weather. This is just the time when a lot of men engaged in active business are very apt to come and tell you that they are not feeling up to their work, that they are feeling a little below par, and that they do not know what to do with themselves. On examination you will find that their arteries are atheromatous, and sometimes you will find traces of albumen in the urine. In many of these cases you will find a large amount of urine of low specific gravity, with the mere traces of albumen which you generally get with gouty kidney and the hypertrophied heart which goes with this. In some you may not find these conditions. On careful examination you may find the murmur over the aorta which is so indicative of atheroma. You may find the vessels are hard, and then you begin to suspect that a good deal of the trouble is due to imperfect nutrition of the brain substance from narrowing of the lumen of the cerebral vessels and atheromatous change. Later on, if care be not taken, these men begin to get little lapses; they may forget to do some important things in their business, they may make mistakes in their business, and sometimes these mistakes may lead to very serious pecuniary losses.

Now, is it of any good to recognise these symptoms beforehand? Can you do anything for them? When you look at an atheromatous aorta you are inclined to say you cannot do very much good there. But if you recognise the symptoms just at the beginning I think you may do a good deal. The process of atheroma is very much the same process as that which goes on in rheumatic arthritis. When you have a man with stiff joints, if you send him to a bath where he gets put through a course of internal washing, and massage to his joints, you often do him a great deal of good. His thickened joints become smaller and more natural, and he can walk about freely. You can apply the same process to a great extent to these atheromatous vessels. You can get the waste products washed out by water taken internally, and you may get massage applied to the vessels by means of gently graduated exercises. You will find a description of the way in which it is done by reference to the pamphlet which I wrote on atheroma, and which I have sent round. But it is very difficult to get a busy, middle-aged man to give up sufficient time to himself to carry this out, and therefore it is better to tell them to go to some place where they can have the exercises and baths systematically given, and which will be combined with fresh air and very likely cure. Care must be taken not to overdo the exercises, and then the patient will be helped with his circulatory trouble, and there will be a satisfactory absorption of the products of the atheroma. Of course, when you get a patient with disease to the extent which this old lady had you cannot do very much for it, except to give the patient a dose of calomel occasionally, as was done in her case.

The Curability of Tuberculous Meningitis.—In a review of the rare cases of tuberculous meningitis in which recovery occurred, Paul Sépet states that in such the process is generally well localised. General tuberculous meningitis is always fatal. He reports two cases in children. Lumbar puncture was only performed in one case. Both children had typical symptoms, and recovered. Meningeal tubercles or other well-circumscribed tuberculous meningitis is therefore curable.—*Philadelphia Med. Journ.*, September 13th, 1902.

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A DEMONSTRATION OF CASES*

IN THE

SURGICAL OUT-PATIENTS' DEPARTMENT, WESTMINSTER HOSPITAL,

May 20th, 1902.

By WILLIAM TURNER, M.S., F.R.C.S.,

Senior Assistant Surgeon to the Hospital, and Surgeon to the Seamen's Hospital Society, Dreadnought Hospital, Greenwich.

GENTLEMEN,—The first two cases I have here for your inspection are both instances of osteo-arthritis of the knee. In some respects they are similar, and in others they are different from each other. In both patients one joint only is affected, and in both there is trouble outside the joint as well as inside. In the man there is a ganglionic swelling which is easily felt in the front of the right side, just above the head of the fibula. The woman has had the same in the popliteal space. You see the woman is very big, although her age is only twenty-five. I saw her first in January last, and her trouble had then dated back six months. Six months before this she began to get stout, and she has been increasing in weight ever since, from 8 st. 2 lbs. to 13 st. 4 lbs. to-day. The trouble in the right knee commenced with pain and stiffness and difficulty in getting about. The pain had been very severe for five months, and varied with changes in the weather, but after that the pain diminished somewhat, although the local signs had increased. When she first came she had what you see now, swelling of the synovial membrane affecting the whole of the joint, and very marked grating on movement of the joint, but in comparison it was much more pronounced then than now. The popliteal space was filled up with a big swelling, which was an enlarged bursa, which is now very much smaller. The main point of interest is her very great improvement on a certain line of treatment, which is the last one anyone would adopt for such a condition, namely, "rest" by means of a plaster-of-Paris splint. I have previously tried the ordinary measures, such as Scott's dressing, massage, etc., but without success. Notice that now the movements of extension and flexion are normal, and this although she has been at rest for ten weeks. The swelling in the popliteal space has gone down; it is nothing like so large as it was, and her condition generally has improved markedly; but she is still increasing in weight. I think we must say we know nothing really about the ætiology of cases such as this; though the fact of a young woman of only twenty-five years of age increasing suddenly in weight from 8 st. 2 lbs. to 13 st. 4 lbs. in a space of eighteen months, accompanied by the fact that she gets such a knee as this, points certainly to some altered condition of general nutrition taking place. But what I wanted to mention specially in connection with this case is the satisfactory result from treatment by rest in a plaster-of-Paris splint. I thought at first it would be a case which would have to come to excision, but her improvement is such that I think she should be put back into plaster of Paris. At one time we thought she might have myxœdema, but we had to relinquish that idea; and she could not take thyroid extract without producing great discomfort and symptoms. She is taking arsenic, which, as a rule, is our stand-by in such cases.

This man presents a similar kind of condition, but it is really quite different in many respects. In him there is apparently the same kind of inflammation of the joint, *plus* this localised swelling about the size of a shilling above and just in front of the head of the fibula. Eighteen months ago he had a severe injury to his knee, and his present symptoms date back twelve months. He has very much pain on attempting to walk, and he cannot get about at all except with very great difficulty. He is forty-one, and he is a person of far different physique from the woman; he is what you may term cadaverous-looking. I think the local condition is of much the same nature as that in the woman; namely, osteo-arthritis. The swelling in front of the joint itself moves on flexion of the knee, and is most marked just in front of the head of the fibula when the knee is extended. I take it to be a ganglion, with a similar pathology to a ganglionic swelling from the sheath of a tendon about the wrist-joint, and if it became detached it would be called a Morrant Baker's cyst. He had a quantity of fluid in the joint. There is no syphilitic history in this man, and no signs of gout or rheumatism. I think it is a case in which operation will have to

* For Post-graduates.

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be undertaken to effect improvement. The first thing necessary is to cut down upon this ganglion, as all his pain seems localised here. The patella is fairly free, but the edges of the bones are not very marked, being covered by the fringes of synovial membrane; but this is not at all like the thickening present in a tubercular joint. His movement is fair, but on account of the pain in the joint on movement he finds it very difficult to get about. In a case like this rest is not suitable, and he has shown no improvement in the last fourteen days. I think the best term for these cases is osteo-arthritis, which I take to be a condition of chronic inflammatory changes in the cartilage and synovial membrane which does not tend to break down, but rather to cause stiffness and ankylosis in the joint, giving rise to pain, particularly when the joint is at rest in the warm, or after exercise. Some few cases remain with a quantity of fluid in the joint; others go on to disorganisation of the joint, with osteophytic growth at the edges of the bone, leading to the lipping commonly seen in later stages. Both these cases are in the early stage. A number of these cases may occur in patients with rheumatic, gouty, or syphilitic diathesis, and in a great many trauma is the exciting cause. I have a great belief in trauma as giving rise to many of these cases. In the case of young persons where many small joints, such as those of the fingers, are affected, I think that though in many respects some of the pathological processes are the same, there is and must be a marked difference in the ætiology. I think some change in the nervous system must enter largely into the cause of such cases.

I want next to show you an old gentleman who is suffering from malignant stricture of the œsophagus under treatment with a Symonds' tube. He is 63 years of age, and I saw him first in January last. His history was that he came up to the Chest Hospital with a very troublesome cough and much frothy expectoration, but no hæmoptysis. When examined nothing was found but a few general crepitations and rhonchi, but as he looked ill he was admitted. On admission this history was elicited:—For the last six or eight weeks he had been unable to swallow solid food; when he tried to do so it appeared to stop at the bottom of the sternum, and was then regurgitated after some minutes. He was able to swallow liquids. His weight was 8 st. I saw him on January 22nd, aneurysm having been excluded. It was found that he could not take anything except fluids. Œsophageal bougies were attempted to be passed, but the smallest—No. 4 catheter gauge—only went twelve and a half inches from the incisor teeth. Six inches from the teeth brings us to the commencement of the œsophagus, and the œsophagus is nine inches long, totalling fifteen inches, so it will require sixteen inches for the bougie to get through to the stomach. Twelve and a half inches brings the tip just below the bronchus, or just above the position of the lower border of the root of the left lung. A large size bougie (No. 16 catheter gauge) was stopped at ten inches. The question was what to do for him. I could get nothing through the stricture. I discussed the matter with him, and he begged not to have gastrostomy done. So he was kept quiet in bed, fed on nutrient enemata, and only allowed albumen water per mouth; and after well cocainising the œsophagus by means of cocaine lozenges I was able on the 29th, one week later, to pass a whip, and then the smallest size Symonds' tube over it.

This tube answered until February 17th, when it had to be removed, as the patient had been unable to take any food through it for twenty-four hours.

On the 22nd the stricture was dilated by bougies up to No. 14 catheter gauge, and a No. 10 Symonds' tube introduced.

On March 10th this tube had to be withdrawn, and on the 12th all bougies were found to pass only ten and one-eighth inches from the incisor teeth. With difficulty over a vulcanite whip, a No. 15 Symonds' tube was introduced, but the patient suffered much pain, and there was considerable amount of hæmorrhage; the pain was so great that for the following twenty-four hours he had to be fed on nutrient enemata, as he was unable to swallow; after that the tube worked well.

He was discharged from hospital with a No. 15 in on April 2nd. On April 26th the tube blocked again, and had to be removed. On April 28th No. 15 tube was again introduced, and glands were noticed becoming enlarged in supra-clavicular fossa.

This tube is still in, May 20th, and working well; and he is able to soak biscuits in milk and take them with eggs beaten up. I show this man

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to you to illustrate how closely the results of treatment by Symonds' tube simulate those by gastrostomy.

The patient must have the silk attached to the tube hanging out of his mouth, and he puts that round his ear. The history points definitely to a growth which is spreading upwards, and the physical sign which definitely tells one that the stricture is not of a simple nature is present, namely, the enlargement of the glands in the neck and supra-clavicular region. Some people may want to know why I did this in preference to treating him by gastrostomy. The reason was that I put the whole position plainly before the patient, and asked him which I should do, and he begged not to have the operation performed. Now, shortly, what are the advantages and disadvantages of this method of treatment?

The advantages are surely that the patient can take food, though liquid, through the mouth, and does not have to partially undress to feed himself through a gastrostomy tube, and he does not run the risk of the operation or any of its after discomforts, as eczema, etc. Again, having introduced one tube, a larger size will nearly always be able to be passed in a short time, and so increase his facility for taking food.

The disadvantages are certainly numerous: first, the discomfort of having instruments passed is great; then there is undoubted risk of hæmorrhage, which may be great, and also perforation of the growths may occur. It seems also possible and probable that the presence of the tube may determine the extension of the growth (1) along the œsophagus, as has occurred in this case; and also (2) away from the lumen of the tube into surrounding parts, as the aorta and the lung; but these same complications occur in cases not treated by this method.

The average duration of life in these cases seldom exceeds six months, and four months is the more common: this patient has now been under treatment four months, and can get about and manage the affairs of his daily life. In hospital he increased steadily in weight, about 1 lb. a week when the tube was working well, but since leaving hospital he has gone down again a few pounds; but the alteration in weight only occurs very slowly.

The upward extension of the growth is shown by the shorter distance the bougie is able to be passed each time.

I want to show you next this old man, because he has got a condition which, though common, is seldom seen. He has a dislocation of the hip on to the dorsum ilii. I have met many medical men who have never seen such a case. He is a sailor, æt. 62, who fell down the hold of his ship while serving in the Mediterranean. His head was also injured by the same fall; the condition was not diagnosed, or if diagnosed, attempts at reduction failed, and four months after the accident he presented himself at the Seamen's Hospital, and the condition recognised. You see there is marked shortening, and distinct adduction of the left thigh, but there is no inversion, but when first seen that leg was very much inverted. When he walks, as you will probably have noticed, he keeps his foot exactly straight forward. Notice the relation of the trochanter to the anterior superior spine. Bryant's line on the affected side is very short indeed. Notice also the ischial tuberosity on both sides, and the head of the bone lying on the dorsum ilii above and external to it on the affected side. I have given him an anæsthetic four times, and have tried to manipulate the bone into position,—in fact, four of us have tried, but without success. I have never used so much force in a case before. I have also tried with pulleys, but again without success. You must remember he did not come under treatment by us until four months after the accident, which is a very long time. All that the manipulations have done is to enable him to evert the leg; that is to say, we have freed the part around the head of the bone. But he notices when he stands the want of support for the head of the bone. You see he is a heavy man. Can anything else be done? Well, there is only one other thing, and that is to cut down upon it. He is sixty-two, and it is nearly five months now since the accident, therefore if I were to cut down, in what condition should I find the acetabulum? It is nearly certain to be nearly or partially obliterated, and even if the dislocation was reduced, ankylosis is very likely to follow, and then will he be better off than now? I think not. Whatever we do for him it is certain he cannot do the ordinary work of a sailor again. But if he had been a younger man I should not have hesitated about operating on him. His walking powers are,

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I am glad to say, improving gradually. There is another point, and that is, if he had been young, I should probably have been able to reduce it.

Next I have a very interesting chest case, and I brought him to show you three or four unusual features. His history is very interesting. In July, 1898, that is to say, three and a half years ago, he was admitted to the Chest Hospital. His age was then 17. Towards the close of June, 1898, he had a shivering attack while in bed, followed by a stabbing pain in the right side in the axillary region. But he continued his work, and saw no doctor until July 19th, when he fell down at work from a sudden acute, severe pain on the right side. On admission his temperature was 104·2°; respirations 30, short, shallow and jerky; pulse 160, and he complained of very severe pain in the right side.

The right side of the chest in front and behind was dull; breath-sounds were absent. V. F. and V. R. diminished; the apex-beat 1½ inches external to the nipple line. Liver dulness 2 inches below the margin of the ribs. A needle was introduced into the eighth space behind, and turbid fluid and air was withdrawn. Relief followed, but re-accumulation necessitated aspiration daily until the 26th, when the fluid obtained was found to be purulent. The later aspirations were performed through the third space in front 2 inches above the nipple. This spot was chosen as being the position where access to the air in the pleura was easiest. It is a very unusual position, and has the great fault of the needle having to penetrate the pectoralis muscle.

I saw the patient on the 26th, and under A.C.E. removed 4 inches of the eighth rib in the posterior axillary line, and made a large opening into the pleural cavity, 3½ pints of pus being evacuated, containing all descriptions of micro-organisms. This completely healed, as you see, and no ill-effects have followed from the periosteum of the rib being left at the operation.

It is sometimes stated that the periosteum must be removed when draining the pleura. This operation was done three and a half years ago, and bearing that in mind I think you will admit the result is excellent, although some new bone has been formed, and gives the patient no discomfort. I never do remove the periosteum in such cases. On August 5th—that is to say, a fortnight after this operation—I went down to see him in response to the house surgeon's summons, and I found he had a fluctuating swelling in the position corresponding to the site of the aspiration, deep to the pectoral muscles. Being under the pectoralis major I saw that it had no chance of draining through a direct opening over it, so I made an incision at the outer margin of the pectoralis major muscle, and drained the abscess at the side. This was followed by some superficial necrosis of the fourth rib. He is perfectly well as regards this side, and I had lost sight of him, but he was re-admitted for hæmoptysis seven weeks ago, and developed a few crepitations at the base of the left lung in the anterior axillary line. There are many points of interest in this case: first, from the history, there is no doubt that there was a rupture of a small phthisical cavity, and that cavity contained not only tubercle bacilli, but septic organisms of the ordinary type. Secondly, the communication with the air-passages was covered by a valve, so that air could enter, but was unable to get out of the pleura. Thirdly, the position in which the aspiration was done was a very unusual one, and was only chosen on account of the physical signs being most marked there, and was followed by an abscess superficial to the ribs. The cases on record in which an abscess has followed the introduction of an aspirating needle are rare, but it is mentioned in all books as occurring, and as one of the dangers to be avoided if possible. Fourthly, the patient had an attack of hæmoptysis a few weeks ago, and was admitted for it, but there were no physical signs. However, after a week a few moist sounds were discovered in his left axilla. So you see the diagnosis of tubercular disease has been proved. And, lastly, the excellent result to the right side from the operation, even though the periosteum was left intact.

Next I show you four men who present secondary syphilitic rashes in different stages. One of them has, in addition to his rash, rather severe iritis. The first man is quite an ordinary case. The reason of it having been so extensive is that he has had very little treatment for his syphilis; still, one week's treatment has practically annihilated his rash. It is in the roseolous form. You see it is going on to the typical coppery colour. The coppery colour does not usually show itself until after some treatment. He has had a primary sore on the prepuce, which has now practically

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gone, but the glands in the groin are markedly enlarged. In secondary syphilis, in order to clinch a diagnosis I generally look for epicondylar glands. He has a very severe attack, and I gave him large quantities of mercury and a mercurial ointment to rub in; and, in fact, an over-dose, and produced symptoms of mercurialism. There has been stomatitis, and great redness of the gums, especially around the teeth, with that dirty tartarous condition of the mouth generally. Still, the disease was practically stopped for the time being. The rash has hardly become papular yet. I propose for him to stay on mercury for fifteen to eighteen months. It is six weeks since the primary infection, so you see it is a very rapid case altogether. You see it has been very easily amenable to mercurial treatment. Passing on we come to three men, who are all cases of secondary syphilis in a later stage; they are all sailors, attached to sailing ships. The first man has been under treatment five weeks; he became infected five months ago,—that is to say, on Christmas Day. He went to sea with the sore, and the rash commenced to break out four days afterwards. As he had no treatment his case ran on to May, so that he went four months without treatment. That makes nearly six months from the time of the original inoculation to the time of the development of these rupial sores. You will notice that some of the scars are keloidal. Nearly all the ulcers have healed now. In the next patient there is a true papular rash. The papules have gone on and broken down and become vesicular, and in some parts they have ulcerated. There is, especially on the arms and legs, an impetiginous variety going on with the local suppurative infection. You see the site of the primary sore, which has practically healed. In the next man nearly the whole of the rash is papular, which has gone on to true rupia. In places many of these papules show umbilication. He has no macular rash worth mentioning. His is a much longer case, and it dates over six months. Here is the seat of the primary sore, but there is no induration about it. He says he came out in pimples three months after he got a chancre. You will notice also some patches of rash in the psoriasis stage. In this man you can feel the epicondyloid gland rolling about under your finger on both sides. These glands very rarely get enlarged in any other condition; so that when you find it enlarged on both sides you can almost be sure the patient has got secondary syphilis. It is occasionally enlarged in tuberculosis and lymphadenoma, and from septic sore on the hand; but in these later cases the glands in the axilla are usually more likely to be affected. In this last case there are some regular subcutaneous nodules. It is early cases of this description which get mistaken for smallpox. Quite a number of these cases get sent to the smallpox hospitals. This last man has also a very acute iritis, with a great mass of gummatous granulations on the outer side. The great point in the treatment of these cases is mercury; but it is also necessary to give the patients good food, rest, and no alcohol. It must be remembered that cases such as these come off sailing ships often in a semi-scurvy condition, after having been perhaps weeks or months continuously on board. I have only seen one case in which it was impossible to give mercury at once, and she had acute symptoms after 1 grain was administered. We treated her by cod-liver oil, iron, quinine, arsenic, strychnine, and port wine. Her lesions got well, but I do not know that we have cured the syphilis by that means, and unfortunately I have lost sight of her.

I hoped to have shown you a young woman with secondary syphilis, who shows very little evidence of it, and illustrates very well the fact that women often do not manifest the disease anything like to the same extent as men. She has sores round the vulva, which are not characteristic, enlarged glands in the groin, and two mucous patches in the mouth on the tongue. But she has no sore throat, no rash, and no risen temperature.

It is cases like this that lead afterwards to anomalous symptoms with no definite history of a previous attack of secondary syphilis.

Yeast in Therapeutics.—Paschkis has used yeast in about twenty cases of furunculosis, acne, or severe folliculitis, and found that its therapeutic action was remarkably effective in some of the cases, while others proved refractory. Brocq claims that it is as much of a specific in furunculosis as quinine in malaria or mercury in syphilis. There are no inconveniences from its use, except that possibly if much sugar is being taken at the same time, the fermentation set up might induce considerable flatulency.—*Journ. A.M.A.*, September 13th, 1902.

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The Index and Title-page for Vol. XX will be published with the next issue of THE CLINICAL JOURNAL.

A CLINICAL LECTURE

ON

ADENOIDS.

Delivered at St. Bartholomew's Hospital, July 14th, 1902.

By ANTHONY BOWLBY, C.M.G., F.R.C.S.,

Lecturer on Surgery and on Diseases of the Throat.

GENTLEMEN,—You know that the term adenoid growth or "adenoids" is a term which is applied to overgrowth of glandular or adenoid tissue at the back of the pharynx. This condition, which is exceedingly common, is, after all, only an exaggeration of the normal condition, in exactly the same way as enlarged tonsils are an overgrowth of the normal condition. You cannot too clearly keep before your patients the fact that this is not a new thing; that it is not something growing there which should not be there at all; you should give them to understand that in exactly the same way as the adenoid or glandular tissue of the tonsils may overgrow, so also may the adenoid tissue of the pharynx overgrow. That is the first thing which it is necessary to appreciate in order to understand what adenoid growth implies. The next thing to consider is, if it is only an overgrowth, where and what is the normal growth? There is normal adenoid tissue in the back of the pharynx in every newly-born child. This is collected more or less into a definite mass in the pharynx, and goes by the name of the "pharyngeal tonsil," a good name in so far as the structure of this adenoid enlargement is of exactly the same structure as the normal tonsil, so that the section of one placed under the microscope cannot be distinguished from a section of the other. This pharyngeal adenoid tissue exists on the posterior wall of the pharynx as a normal condition, and it extends up towards the roof of the pharynx; but it is not found anywhere else. There is no adenoid tissue around the orifices of the nostrils, on the palate, on the pillars of the fauces, or at the end of the Eustachian tube; and therefore

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that which you sometimes hear about a collection of adenoid tissue at the orifice of the nostrils and so on, is misleading. It is true there may be a great deal of swelling in these parts in patients who are afflicted with overgrowth of adenoid tissue, but this swelling is secondary; it is due to catarrh. This swelling of the orifice of the Eustachian tube is entirely analogous to the swelling which may occur further forward in the nose, and which results in overgrowth and enlargement of the turbinated bodies. But nobody would suggest that the enlargement of the turbinated body is due to a growth of adenoid tissue; it is a swelling due to congestion.

So much as to what adenoid growth is. Now I want to talk very briefly about the symptoms it causes. I think they can be most conveniently put into three classes—nasal, aural, and pharyngeal. The symptoms due to adenoid growth are not the result merely of the size of the swelling, any more than all the symptoms of enlarged tonsil are the result merely of the overgrowth of the tonsil. There may be a considerable overgrowth of tonsil, with next to no symptoms; or there may be only a small tonsil, which may become the source of a great deal of trouble. The same is true of adenoid growth. It may attain a very considerable size, and yet it may give rise to no trouble. Whether it will give rise to trouble depends on the extent to which it is associated with or causes catarrh. That is the key to the position, and in many cases you will find that children who have evidently had adenoid growth for some time have no trouble, until, for some reason, perhaps as the result of an illness, there is set up a catarrh, which catarrh continues so long as the adenoid growth remains. The symptoms which you have to look to are those which result from an extension of the catarrh first of all towards the nose. The nasal cavities may be partly blocked by the mere size of the large mass of adenoid tissue. But they are much more often blocked as a result of the extension of the catarrh and the presence of nasal catarrh, which is liable to exacerbations, so that the parents will often tell you that the child is constantly having colds in its head. They mean that the child is constantly suffering from nasal catarrh. It is this catarrh more than the mere lump of adenoid tissue which gives rise to one of the characteristic symptoms of adenoids, namely, nasal obstruction; and thus nasal obstruction is a common result of adenoid growth. But, while these nasal symptoms are very common—so common as to be looked upon as the commonest symptoms in connection with adenoid growth,—they may be entirely absent.

Aural Symptoms.—Next we will consider that the catarrh may spread in the region of the Eustachian tube, and up the Eustachian tube, and towards the ear into the tympanum. As a result of this extension of catarrh, the main symptoms may be associated with the ear, so that the child may be said to be a little hard of hearing; or that it has attacks of deafness; or that it has occasional ear-ache; or that every time he gets a cold he becomes deaf, and that the deafness lasts longer each time. These are largely the symptoms due to a catarrh spreading to the Eustachian tube, and if the mischief be not dealt with, and nothing be done, there will ultimately be an extension of this to the cavity of the tympanum, and there will be temporarily tympanic catarrh. Ultimately, at a time when there is a more acute inflammation of catarrh, the mucus in the cavity of the tympanum, unable to get out, may distend the cavity of the tympanum and produce severe earache, and, a muco-purulent catarrh following, the membrane may be perforated; this perforated membrane will not heal so long as there is the Eustachian obstruction. Exactly in the same way that a fistula of the perineum is not expected to heal so long as there is a stricture, so in the same way if the Eustachian tube is obstructed and there is a hole in the membrana tympani, you must not expect that hole to heal up. If you were to set to work to cure a urethral fistula you would naturally first try to cure the stricture. You must do the same here. Perforation of the membrana tympani in patients who have adenoid obstruction and catarrh resulting from it, must be treated by removal of the cause. Next, you must remember that the ear symptoms of adenoids may be present alone. There may be no nasal catarrh, and there may be no other symptoms of adenoid growth; the catarrh which has been set up may spread only towards the ear. Therefore, you must be on the look out for cases where, in children, there are no symptoms of the commonly recognised class of adenoid growth being present, but where the child is deaf. You may ask for symptoms of nasal obstruction. There are none. The patient's mother will tell you the boy breathes

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well through the nose, that he has no nasal catarrh, and of that you can perhaps satisfy yourself; nor is there any evidence of nasal obstruction. Therefore, remember that in some of these cases the deafness may be the only symptom of the growth of adenoid tissue within the pharynx, and of the resulting catarrh. I remember very well some years ago seeing a little girl aged five, who had steadily been getting more deaf for a year. She had no symptoms of adenoids at all except deafness. Her tonsils were not enlarged, she had no difficulty in breathing, no nasal obstruction, and she slept quietly and well. I was at first very disinclined to operate upon this child for the possible adenoid growths which we knew might be there, because she had no other symptoms. But after treating her for three months, and satisfying myself that she was getting worse, and finding she had retracted membranes, I removed what turned out to be a very small quantity of adenoid growth from her pharynx. In ten days or a fortnight after that operation her hearing was completely restored, and she has remained well ever since. You have also probably seen in the throat department, or you can see, plenty of patients who are sent over from the aural department because they are deaf, and often without any other symptoms. And it is my constant custom, under those circumstances, to proceed to at once operate upon any adenoid growth there may be, because in cases of this kind, where there is deafness in a child without any other obvious cause, there may be adenoid growth to account for it, though there may be no symptoms specially pointing to the adenoid growth. Ear symptoms, therefore, may co-exist with nasal symptoms; or ear symptoms may be alone present in the case of adenoid growths of the pharynx.

Pharyngeal Symptoms.—Thirdly, there are the pharyngeal symptoms. I have alluded to nasal symptoms and aural symptoms, and now we will consider the pharyngeal ones. The pharynx is the seat of this growth, and therefore you would expect to find some symptoms referable to the pharynx and to the catarrh which is present in the pharynx. And were it not that your patients were so often young, and that they have been accustomed to this pharyngeal catarrh for so many years in early life, they would complain more of symptoms than they do. As they grow older, these symptoms are complained of, but in childhood, as the patient is used to them, they become natural to him, and there seems to him to be nothing abnormal. But when patients get older they complain of pharyngeal mucus, or of something which they are constantly feeling is accumulating at the back of the throat, and they have a difficulty in clearing it away. This is a result of the catarrh of the pharynx. But though the child does not complain of this, there are other evidences of pharyngeal catarrh, in the presence of a cough or of enlarged glands in the neck. These pharyngeal symptoms also may co-exist with nasal or aural symptoms, or they may be alone. A child may have trouble in the pharynx of which it can only very obscurely complain, without having any nasal trouble, and without having any aural trouble, and this may be due to the adenoid tissue setting up and maintaining catarrh. Some years ago I saw a small boy who for several years had constantly suffered from a short, hacking cough. He was otherwise well and strong, and had no nasal or aural symptoms. He had been seen by various physicians on account of this cough, and that was the only symptom which he had. He complained of nothing. The cough was suggestive of a permanent irritation in the throat, and without there being any other symptoms to justify it, I advised that the adenoid tissue which he might have should be removed. The operation was done, and from that time onwards the cough was completely cured. Cough of a hacking character in a small child may, then, be associated with adenoid growth, and that, with the accumulation of mucus in the pharynx, and enlarged glands in the neck, may all be taken as evidence of the pharyngeal catarrh. These symptoms may exist by themselves, or there may be symptoms referable to the nose and ear.

Next we have to consider what other further change or changes you may notice of the presence of adenoid growths in the pharynx. One result, which is more or less important, is the alteration in the appearance of the child in its facial expression, or want of expression; and you are probably familiar with the fact that where the nasal symptoms predominate and there is much nasal obstruction, the child assumes habitually the open-mouth appearance, a want of the ordinary natural expression. And, what is not so commonly recognised, is that a result of this constant open-mouth and of the imperfect support supplied by the lips

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to the teeth, is a protrusion of the upper central incisors. Do not imagine that every child who has prominent central incisors has got adenoid growths. But recollect that in a certain number of cases where the open-mouth has been habitually the condition for a long time, as a result of that there may be prominence of the teeth, because of the want of pressure of the upper lip against them, and that a means towards getting the teeth in the natural position, probably with the assistance of the dental surgeon, is to remove the adenoid growths.

So much for expression as regards the mouth. The nose is also liable to be altered permanently. Some people attach more importance to the appearance of their children, especially if they are girls, than other people do; but necessarily the alteration of the face of the patient is a matter of some importance. And you may have one of two classes of nasal alteration in these patients. Either the nose may be very flattened from side to side, with very narrow slit-like nostrils, or it may be thick and stumpy, with a broad base, and consequently ugly. And patients who have had long-continued nasal obstruction in early life may have either of those conditions present. For, if during the period of development of the nose that organ is not used for breathing, the nostrils are not properly expanded and developed, and I think the broad, stumpy nose is the result of the constant congestion of the parts which so often follows upon the growth of adenoid tissue and the constant nasal catarrh. There are one or two more references to the general condition of these patients. Many of them sleep badly and eat badly. One reason why they sleep badly is the difficulty that they have in breathing at night. You know that in normal sleep the nose only is used for breathing, and if the nose only is used by these patients an insufficient supply of air gets to the lungs, so that the child gets more or less semi-asphyxiated, wakes up, struggles, and gasps for more air, and having obtained it, goes to sleep again. Many of these children snore; they snore when they do not breathe through the nose—when there is so much obstruction that the only passage for the air is through the mouth.

I have also said these children are often bad feeders, and one reason is to be found in association with the pharyngeal symptoms. Patients who are old enough to complain tell you that where there is pharyngeal catarrh their trouble is a nasty, sticky secretion in the back of the throat, and it often puts people off their food. Children cannot complain of this; they hardly know how to explain of it, because many a child with adenoid growth and pharyngeal catarrh cannot remember the time when it had anything different; to not a few children it is their only condition. In many instances the poor appetite is due to the secretion of mucus in the pharynx, as the result of pharyngeal catarrh. Taking these circumstances into consideration, it is not to be wondered at that many of these children do not grow properly, so that in a number of these cases the development of the child is arrested, and the health to some extent impaired, and they become weakly and flagging, and have little energy; there is not that flow of healthy spirits which the ordinary child ought to have. Many such children are consequently under-sized. Some of the symptoms to which I have referred may be noticed without there being any of the local symptoms. A child may sleep badly, may eat badly, may develop and grow badly, without there being necessarily anything associated in the minds of the parents with nasal, or pharyngeal, or aural trouble. So you see that the symptoms of adenoids necessarily vary widely, in proportion as to whether certain parts are affected or not. I want you to appreciate that the nasal, or aural, or pharyngeal symptoms, or the general symptoms to which I have alluded, may predominate, or may co-exist in varying degrees. It will be very seldom that you will meet with a single patient who will have all the symptoms I have mentioned; there may be one, or there may be more. In cases where there are many symptoms, and where they are characteristic, you are not likely to be easily misled. But if, on the other hand, you do not appreciate that a child may be deaf, and yet that it may have no other symptoms; that it may have an irritative pharyngeal cough and no other symptoms; that it may have a bad appetite and no other symptoms; that it may sleep unsoundly and be a badly developed child, and yet have no deafness; you are very liable to overlook certain cases that should be treated by operation.

I will not say anything more about the symptoms, except to point out this: that you will find the patients will improve after operation in propor-

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tion as the symptoms are noticed sufficiently early for you to operate at a reasonable stage. If the symptoms have been continuing for an indefinite number of years, your operation will come too late. If, on the other hand, they have been noticed sufficiently early, either by the watchful parent or by the doctor, then the operation will result in a remission of all these symptoms.

Let us take the symptoms one by one. If you operate too late, as far as nasal symptoms are concerned, you will not prevent the patient from having permanently narrowed and small nostrils, and an altered shape of nose. If you operate too late, as far as the aural symptoms are concerned, you will not prevent the patient from having permanent Eustachian obstruction, with chronic catarrh and permanent deafness, which is more or less complete. If you operate too late, you will never completely get rid of the pharyngeal catarrh, which, if it continue for a sufficient length of time, may accompany the patient for the rest of life. And lastly, if you operate too late, as far as the general condition and development is concerned, you cannot expect that if the child has once grown up to be a young adult, that the operation will have any effect upon the stature or development.

Typhoid fever would hardly seem to be a disease to be treated surgically, yet in a recent number of the 'Presse Médicale' Dr. Dauriac, a former house physician of the Paris hospitals, has described an operation he carried out in a severe case of typhoid fever complicated by hæmorrhage. On the eighteenth day of the illness the temperature fell suddenly to below normal, and the pulse went up to 152. The next day the temperature went up again, and the pulse was about 156. Dr. Dauriac was asked by the father of the patient to perform the operation he had spoken of, and which consists in establishing an artificial anus on the small intestine. Dr. Dauriac chose a part of the intestine which was healthy in appearance, and through the aperture made he washed out the lower intestine with two litres of saline solution every day. The patient began to improve almost immediately, and six days after the temperature had gone down to normal. The artificial anus was closed later without any difficulty,—on the thirty-sixth day.—*Therapeutic Gaz.*, September, 1902.

A NOTE ON
SCARLET FEVER
ARISING AFTER OPERATIONS ON THE THROAT.

By the late J. W. WASHBOURN, C.M.G., M.D., F.R.C.P.,

Physician to the London Fever Hospital and to Guy's Hospital.

It is by no means uncommon to find scarlet fever arising after an operation upon the throat or nose. For instance, R. H—, æt. 9, was operated upon on April 16th for the removal of adenoids and tonsils. On April 19th he had a rash, and underwent a mild attack of scarlet fever with subsequent desquamation.

I. S—, æt. 4, was operated upon for cleft palate on June 17th. On June 20th there was a rash, and she underwent a severe attack of scarlet fever with subsequent desquamation.

L. R—, æt. 22, was operated upon on February 27th for the removal of tonsils and adenoids. On the 28th the temperature was raised, and on March 2nd a rash was observed; she suffered from a mild attack, and desquamated.

K. J—, æt. 19, had some adenoids scraped off from the posterior nares on November 9th. On November 12th she vomited, had headache and a rash, and suffered from a mild attack of scarlet fever.

Another case was seen some time ago in a nursing home. The patient had been operated upon for the removal of a portion of a turbinated bone. Shortly after the operation she had what was considered to be a surgical rash, which was followed by marked desquamation. A relative who was looking after her subsequently developed scarlet fever, showing that the original attack was one of scarlet fever.

F—, a child of about 8, had the tonsils and adenoids removed; three days later the temperature was raised and a rash appeared; pulse 130, circumoral ring. When seen with Dr. Hills, of Finchley Road, the case was diagnosed as scarlet fever. Five months previously there had been scarlet fever in the house, but it had been thoroughly disinfected.

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Miss D—, seen with a consultant, in whose case the operation had been performed upon the nasal septum. Two days later a rash appeared, and she had a typical attack of scarlet fever, followed by desquamation.

These cases are of very great interest, and there are two possibilities in connection with them: the one that the operator introduced the scarlet-fever virus on his instruments or fingers, and the other that the virus was present in the throat at the time of the operation, and that it gained entrance through the cut surface. The period at which the rash came out in almost all the cases shows fairly definitely the causal connection between the operation and the attack. The case in which scarlet fever had previously been in the house suggests that the child had the virus in its throat ever since the occurrence of the scarlet fever. When one remembers that pathogenic bacteria, such as the diphtheria bacillus and the pneumococcus, are frequently found in the mouths of healthy individuals, there is no reason why the virus of scarlet fever should not also be present. Indeed, if this were so, it would explain a great many of the cases of scarlet fever in which the source of infection cannot be found.

WITH DR. DONALD HOOD
IN THE WARDS OF THE
WEST LONDON HOSPITAL,
JUNE 9th, 1902.

Recurrent Typhlitis.

THIS little boy was admitted a few days ago for slight abdominal trouble. He had pain in the right iliac fossa, and there was a history of his having been ill for a few days previously. And, what is far more important, there was a history of his having had four or five similar attacks in the preceding twelve months. On examination a small tumour was found occupying the position of the iliac fossa. On going carefully into the case, and eliminating all other possibilities, it became practically certain that it was a case of recurrent appendicitis. As you see him to-day the child has practically nothing the matter, but if you make deep pressure you will be able to feel a lump, which might be a gland or some fæcal matter, or it may be, as I think is very probable, the remains of the inflammatory condition round the appendix. This is an important case, because it really represents a very large class, and a class of case which we have not sufficiently recognised until the last few years. The older members of the profession who can look back forty or fifty years recognised at that time a condition which was spoken of as typhlitis or peritonitis, an inflammation round the cæcum or appendix, now commonly known as appendicitis. I need not go into the history and symptoms of these cases. We know them well, but I do not think that until within the last few years the extremely mild nature of some of these conditions of relapsing appendicitis was appreciated.

Some two or three years ago a young fellow was brought to me from a public school with a history of having had attacks of what was called in the school "biliousness," for which he was dosed, kept in a day or two, and then allowed about as usual. He was brought to me for my opinion as regards the treatment of these constantly recurring bilious attacks. He was a strong, finely made young man of eighteen. As regards his organic health I should say there was nothing wrong; he was perfectly sound. On examination of the abdomen I could detect no lesion. My opinion was sought with regard to the advantage or disadvantage of treating these bilious attacks with aperients. He had invariably been treated with such remedies. The case passed from my mind until some six or nine months afterwards, when I was summoned into the country to see the same patient. He was then "cramming" for the army, and he had one of his usual seizures. I found a typical condition of mild appendicitis, a slight swelling in the right iliac region; vomiting; a rise of temperature; local pain; constipation. The whole attack passed off within three days. Seeing this attack in its acute stage at once suggested a cause for the previous illnesses. I advised the removal of the appendix during the quiescent stage. Another nine months passed, and I again saw the patient with his own medical man. Again there was a very slight attack of inflammatory mischief in the region of the appendix. It speedily passed off, and within ten days of my seeing him Sir Frederick Treves operated. The

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appendix was found thickened, the lumen destroyed, and a small circumscribed purulent mass at its apex. The whole series of illnesses was explained on the supposition that this young fellow at school had suffered from recurrent appendicitis, the attack extending over three or four years.

Now this is a class of case which is apt to be passed over because the symptoms are so often only those of gastric disturbance. I have seen a large number of them, and almost invariably mild cases of recurrent appendicitis have been treated or considered in their initial stages as merely instances of hepatic or gastric derangement. Are these mild attacks more liable to recur—more liable than those of severe type? Looking back on my experience, it has certainly been the case that when there has been very marked extreme symptoms the tendency to recurrence has not been so great. The patient before us will be placed under my surgical colleague.*

Inflammatory Conditions of the Chest giving rise to Abdominal Symptoms.

Here is a young man who was admitted with orchitis following gonorrhœa, and in addition to this trouble in the testicle he had severe gastric disturbance, vomiting, and stomach pain. Of course, it would be easy to account for such a condition in a sharp attack of orchitis; but on being carefully examined it was found that, in addition to the trouble in the testicle, he had inflammatory mischief in connection with the base of the right lung. The affection was slight but well marked. If you care to listen you will at once detect moist sounds over this area of lung. The point I wish to urge upon you is this, without going into detail. The case is one in which the disease for which he was admitted was diaphragmatic pleurisy, and the pleurisy was ushered in by very marked abdominal symptoms. This patient represents a very importantc lass of case. The subject is so important that only two or three weeks ago one of the medical journals had an annotation to the effect that such cases were not often recognised. The annotation was based upon a paper read before one of the medical societies of London. The writer of the annotation appeared to think that it was rare for diseases commencing in the chest, and giving rise to abdominal symptoms, to be correctly diagnosed. I believe it to be a condition far more common than would be expected. It has been well before our minds in this hospital. It is many years ago since I published my first case, and I am constantly going round the wards and pointing out similar cases. They vary much in detail, but it is very important to bear in mind, if you have a patient who is complaining of extreme abdominal pain, with possibly some gastric irritation, such as vomiting and perhaps jaundice, that if you cannot explain the condition by disease in the abdomen, you should examine the chest with more than ordinary care. I have seen a man of sixty years of age who had been in the habit of passing renal calculi. Suddenly seized with pain, from his own point of view he was quite certain that he was suffering from renal colic; but on examining him I found he had a temperature of 103°, with frequent respirations. Here the pain was absolutely and entirely due to acute diaphragmatic pleurisy.

In some of these cases it is difficult at the first examination to be absolutely certain with regard to the inflammatory condition thus arising in the chest. A few hours later there may be direct evidence of pulmonary trouble. A few months ago I saw a very interesting case. It was that of a lady of between sixty and seventy years of age, who for two or three days had been suffering from agonising pain referred to the right epigastric region. Beyond the extreme pain there had been scarcely another symptom. At the time of my visit there was a limited extent of dulness at the right lower base; there was an evident small effusion. I expressed my opinion that the pain was due to some form of diaphragmatic pleurisy, and that inasmuch as the symptoms were those of great pain and some effusion without rise of temperature, the indication was in favour of a malignant cause. It is appalling how very insidious some of these attacks of chest mischief are in connection with malignant disease. In the case of the patient I now refer to, the course of the disease was rapid, and within a few days my diagnosis was verified. A small malignant growth was found in the lower right chest growing from the vertebræ and implicating the spinal nerves. I want you to particu-

* The patient was subsequently operated upon, and the appendix found diseased. He made a good recovery.

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larly bear in mind that some abdominal symptoms may be explained, and are explained by the fact that the inflammation has commenced in the chest. Perhaps the most marked cases are those met with in connection with a perforating gastric ulcer. The ulcer perforates into the pleural cavity, and the initial symptoms of a gastric ulcer may be pulmonary rather than digestional.

Angina Pectoris.

Here is a man the subject of aortic regurgitation, a very well-marked case. Of course he does not come to us complaining of his aortic valves being incompetent, but he is under our care for one of the not uncommon conditions or symptoms which occasionally accompany disease of these valves and severe paroxysmal pain over the chest. His is rather a special case in some ways, inasmuch as he has suffered from this pain for some long time. He tells us he has had as many as thirty-four attacks in a fortnight. Of course this statement will at once make us qualify the term angina. I think it would be right to use the name as an adjective, and say he is the subject of anginous pain. There is an immense difference between constantly recurring pain over the cardiac area and attacks of true angina pectoris. I think in the case before us these symptoms have been largely produced by the life the patient has been leading. If we could look back and adequately study the conditions of heart during the last century we should find, I think, that angina is not as common now as it was formerly, and this is probably to be explained by the fact that the diseases of heart are now so much better treated and managed than they were previously. I can remember the time when the fact of any patient having a bruit was sufficient to warrant the doctor in keeping him absolutely quiet, not allowing any exercises, and hardly any motion. The result of that inactivity was that the muscle-fibres got flabby, there was an infiltration of fat among the fibres, and then, if sudden exertion or motion were undertaken, patients were much more likely to get angina than if they had had more sensible treatment.

In the present day I think perhaps we have gone to the other extreme. There has been a perfect boom in the present day, originating from across the Channel, in the direction of making people who have had heart troubles, functional or organic, perform certain exercises and take certain baths. As far as such cases have come under my observation there does not appear to be the slightest discrimination between the class of disease likely or not to be benefited by this routine treatment; the anæmic fit, with quickly beating heart, having identically the same course to pursue as the man who has suffered for years from mitral regurgitation! But for all that, the treatment of heart affection has improved enormously, and with a ripe experience one is now able to differentiate, and to advise one case to take exercise and another to refrain from doing so. The cases which one comes across requiring exercise are those in which there is mitral regurgitation with almost absolute compensation. And the cases in which you would feel very diffident in recommending exercise would be those of advanced aortic regurgitation. But in the case of the patient before us with well-marked incompetency of his aortic valves anginous symptoms have constantly recurred. I believe this pain to be largely due to want of proper regulated exercise. He has been treated for his bruit and not for his disease. You will perceive that he is a big, powerful, muscular man. The apex-beat is but little displaced; the pulse regular, high in tension, well sustained, and scarcely influenced by position. He has a good appetite, and is well nourished. His anginous pain is, I believe, due almost entirely to high tension and overloaded portal system.

There are other conditions besides the murmur to which your attention should invariably be directed, the most important being the character of the pulse and the size of the left ventricle, as measured by the position of the apex-beat. In the present case the heart appears to be fairly normal in position; the tissues of the heart seem sound and healthy. I also think that the trouble at the valves is not of a very gross kind. So my impression is that a patient of this class would be benefited by a little gentle exercise and moderate occupation. He is an engineer by profession, and works an engine in which there is much vibration and heavy work, and therefore that occupation is very unsuitable for his condition. I hope he will be able to get some employment which is better suited for his ailment, and I think he will be all the healthier for occupation and exercise. He has been in the habit of taking

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nitrite of amyl very constantly, but I have strongly urged him to discontinue its use. In its place I am giving sulphate of magnesium and gentian, that is trying to relieve the excessive tension. He has been under treatment for about ten days, and has had only one attack, and tells us that he feels better in every particular.

The last case I saw in private was a man who came to me three years ago for debility following acute rheumatism. I examined him with the greatest care, but could not find anything wrong with his heart. He came to see me a few days ago—I had not seen him in the interval. He said, "I want you to overhaul me, I am so short of breath." I did so, and was horrified to find the heart apex three inches out of position, and a most intense diastolic murmur. Diastolic murmur is of far more grave significance than a systolic. He had a collapsing pulse. He asked me about exercises, about going to a spa. I believe Nauheim to such a patient spells death! You should be extremely careful about advising exercises. This patient was a good golf player, and the point I had to discuss with him was whether he ought to continue to play. Now I think golf is one of the worst forms of exercise for a patient with aortic regurgitation. I would rather such a man bicycled. I remember a college friend of mine in 1869 who was doomed on account of his aortic incompetency. For a few years he lived a quiet life, thinking every day his end would come. But he has lived on. His general health is absolutely good, and I think he now no longer looks upon each day as likely to be his last. But his to-and-fro bruit is as marked as it was thirty years ago.

There is another patient of mine living, a barrister and graduate of Cambridge, who came to me twenty years ago. At that time I thought he had as bad an aortic trouble as a man could have, but instead of being dead, he is now doing a big practice at the Bar. I instance his case as showing how these patients may live on. I should not like to state it as always absolutely true, but it often happens that the louder the bruit the less cause for anxiety. The cases which trouble me most are those in which there is but a slight bruit, but where other symptoms of cardiac degeneration are present. For instance, cardiac dyspnœa occurring in a patient who has no bruit may be a symptom of the greatest gravity.

Alcoholic Pneumonia.

I only saw the next patient for the first time half an hour ago. It is a case of pneumonia affecting the right lung. It is well marked. I do not think there is any particular point in the case beyond the fact that the patient has a low temperature. He has been ill about ten days, and he is now more or less delirious. His history is that he is a coachman; he was formerly in private service, and now he is in a jobber's yard. You will observe that there are stigmata on the face. A large area of lung has been involved. All over his chest you will find there are mucous râles. His sputum is frothy and watery, it is not that sticky stuff which one gets in a characteristic lobar pneumonia. The low type of the disease would lead me to hazard the opinion that it is a form of alcoholic pneumonia. These cases always require a little different treatment from those of acute lobar pneumonia. They require more stimulation; they are frequently fatal, owing to sudden heart failure. I have ordered a favourite drug of mine in such atonic conditions—tincture of serpentaria combined with tincture of bark. You have to be on your guard with these cases, you have to be prepared for sudden heart failure. If such trouble should occur do not lose time, but give subcutaneous injections of strychnine and inhalations of oxygen without delay.

Here is a boy æt. 15 years. A week ago he met with an accident, falling from a height. Within a few hours of the fall he began to suffer pain in the right chest. Four days ago the pain was very severe, in consequence of which he stopped work. He had had no previous illness. There is nothing of moment in the family history. The condition on admission was one of great pain and distress in breathing. On examination his chest was evidently the seat of a large effusion; there was fluid on both sides, but more on the right than the left. The heart was much displaced, being pushed upwards more than to the left.

Within a few hours of admission the right chest was aspirated, and about ten ounces of blood-stained fluid was removed. The operation gave immediate relief, and now there are practically no symptoms; the boy expresses himself as feeling perfectly well, and he leaves the hospital to-morrow. You will note that there is still a decided want of resonance

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at both right and left base. The heart being somewhat displaced, I should prefer keeping the patient for a few days longer under observation, but he is very anxious to go home.

There are many points of interest, and I may add of doubt, when discussing the nature of the conditions which are found. The lad declares that up to the hour of the fall he was in perfect health. The pain and inconvenience in breathing followed immediately upon the accident. He was brought to the hospital, and the physical condition I have briefly described here easily found.

It is almost impossible to believe that a lad could be working with a double pleural effusion and yet feel absolutely well! We have no history of any acute inflammatory attack. Insidious or ingravescent pleurisy may come on in conditions of ill-health without an acute attack, but such is negatived by the history. There is another possibility which one has to consider. Is the case one of sarcoma?—a rapidly forming malignant growth, dislocating the heart and producing effusion. If such had been the cause, and the fall but a coincidence, I do not think recovery would have taken place so rapidly as it has done. I think it is an example of one of those very rare conditions of pleurisy which occasionally follow shock or accident. I have only seen two or three, but I know that a sudden shock will produce or determine an attack of pleurisy. You all know how extremely common are adhesions of the pleura, possibly the unconscious muscular effort accompanying a sudden accident may tear such adhesion and set up a subsequent pleurisy. I have no doubt that sudden movement will determine a severe hæmoptysis, but a few days ago such a case came before me. The patient was a young man who had been at one of the open-air "cures" for tuberculosis for some months, and had left as being well. Previous to the "cure" he had suffered from two or three attacks of hæmoptysis. Some months after he had left the sanatorium he was driving a dog-cart, when a dog rushed out of a cottage door, he turned round suddenly to hit it with his whip, and almost immediately was seized with an attack of hæmoptysis. The hæmorrhage was undoubtedly determined by the sudden movement.

WITH DR. E. W. AINLEY WALKER AT GUY'S HOSPITAL.

General Pathology of Inflammation.

In the latter part of the preceding lecture we considered the nature and causation of the inflammatory exudate, and the factors which affect its character and quantity.

Now if the number of leucocytes present in the exudate becomes so great as to convert the clear or merely blood-stained fluid into a thick and slightly viscid opaque liquid we are accustomed to speak of the condition as one of suppuration.

Suppurative inflammation when arising naturally is *always* due to the presence of micro-organisms, though artificially it may be brought about by certain other means. And Simon more than thirty years ago arrived at the conclusion that all naturally occurring inflammations are more or less connected with infective processes. In this conclusion he foreshadowed the later theory of Hüter, of which I shall have to speak directly. Then it was shown by Lister that suppuration of *wounds* is always due to the entrance of infective matter from outside—a discovery which enabled him to lay the foundation of aseptic surgery on a firm and scientific basis.

Burdon Sanderson, also, in 1873, concluded from a series of experiments in which he had endeavoured to produce, firstly, infective, and secondly, non-infective inflammations, that the degree of infectiveness of an inflammation depends entirely on the number of micro-organisms present, and showed that whenever metastatic inflammations occur they are associated with the presence of micro-organisms.

It was then held by Hüter (in 1878) that inflammation *only occurs* when pyogenetic micro-organisms are present in the lesion; and this view has been supported by some other surgical pathologists who have endeavoured to limit the application of the term to cases where at any rate a majority of the so-called classical signs are present and are associated with suppuration. They claim, for instance, that the aseptic healing of wounds occurs without an inflammatory reaction. This is a *reductio ad absurdum* of the whole position. It is, as Professor Adami has justly pointed out, to confound the inflammatory reaction with

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the process of pyogenesis. It is quite impossible to draw a distinction of this arbitrary nature; for, as we shall see directly, even suppuration—pyogenesis—may occur entirely independently of the presence of bacteria; and, on the other hand, there is no sharp distinction either in the immediate *cause* or in the *progress* of the process between a simple inflammation and an inflammation which goes on to pus formation. The differences, such as they are, are only of *degree* and not of *kind* between the two, and the one merges gradually into the other without the existence of a clear dividing line. If a perfectly aseptic incision be made, and the wound surfaces be promptly brought together, an inflammatory exudate at once occurs, and the formation of so-called "lymph" or fibrin seals the surfaces together long before any regenerative process is discoverable in the tissue cells. And this deposit of fibrin on the wound surfaces is brought about by the disintegration in the exuded fluid of leucocytes which have escaped from severed vessels, or have passed out by diapedesis as in any other inflammation It should, moreover, be clearly borne in mind that such an injury as here occurs is not a simple mechanical injury comparable to those which we discussed last week. For here we have, even in the most favourable case, a chemical injury or irritation from the exposure of the severed tissues to the air, which of itself, as we have seen, is quite sufficient to induce the inflammatory process; and in the vast majority of operations there is besides the irritating action of swabs and instruments and sterile water, if not of actual chemical irritants in the form of various antiseptic fluids.

Even the formation of pus, in which the definition of Hüter found the criterion and characteristic sign of inflammation, does not require the presence of micro-organisms for its production by experimental means.

It is now nearly twenty years ago since Cohnheim first investigated the power of various liquids in producing inflammation. He used the following method. The liquid to be tested was enclosed in a sealed capillary tube; this tube was introduced beneath the skin of an animal through a tiny puncture, and was pushed along for a certain distance in the subcutaneous tissue. Some days later, when the skin puncture was soundly healed, the little tube was broken subcutaneously. All chance of a microbic infection was thus avoided, and the uncomplicated action of the substance introduced could be well observed. This method of producing inflammation by the use of what came to be called a Cohnheim's tube has been extensively employed, with the result that it has been definitely proved—especially by Grawitz and de Bary (Strauss, Leber, Councilman) among others—that various chemical substances are capable of producing suppurative inflammation, and notably mercury salts and silver nitrate among the inorganic substances, and castor oil and turpentine in the organic series. These two observers also showed that cadaverin, an alkaloidal substance obtained from decomposing matter, has an inflammatory action on the tissues, and Koch has proved the same fact for tuberculin obtained from tubercle bacilli.

Suppuration from the action of such chemical substances as I have mentioned is a purely artificial pyogenesis. It can never happen under natural conditions because there is no natural means by which these powerful irritants can gain access in concentration to the tissues without at the same time entrance being afforded to pyogenetic micro-organisms. The experiments are, however, of great importance as tending to the conclusion that the local accumulation of leucocytes in suppuration is the direct effect of the action of a chemical irritant.

It follows then from all that I have said, that inflammation is *not*, as Hüter held, dependent on infection. Nevertheless infection plays a most important part in many inflammations, and is the invariable exciting cause of naturally occurring suppuration.

It was at first believed that any micro-organism might cause inflammation. This view, however, was speedily disproved by the discovery that ordinary air germs and water germs were quite innocuous. The presence of particles in air was proved by Tyndal. He showed that the luminosity of a beam of sunlight passing through air is due to reflection from the surface of suspended particles, and many of these particles were found to be living micro-organisms. Similarly it was discovered that numerous bacteria exist in water. But it was found that air could be passed through the subcutaneous cellular tissue of the rabbit, or ordinary water introduced into its peritoneal cavity, without

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producing any inflammation. It followed that the ordinary air and water germs were not phlogogenetic micro-organisms,—that is to say, inflammatory agents. Many of the phlogogenetic bacteria may and often do occur in air and water, especially in the neighbourhood of towns and human habitations, but that is quite another matter altogether.

One of the earliest steps in the direction of establishing the specific nature of pathogenetic micro-organisms was taken by Ogston, of Aberdeen, in 1881, in an investigation as the result of which he made the statement that acute abscesses always contain what he called cluster cocci, now better known by the name staphylococci. And he concluded that the cluster coccús was the specific causal agent of such abscesses.

Koch's great discovery about this time, of culture methods and media suitable for the artificial cultivation of bacteria, provided means for accurate investigation of their properties and character, and with the application of his well-known postulates for specificity has led to the determination of the specific causal agents in many different forms of inflammation. The first book published on the subject was that by Rosenbach, of Tübingen, in 1884.

Already in 1876 the discovery had been made by Burdon Sanderson that from the spreading edge of erysipelas can always be obtained an exudation fluid full of cocci which are arranged in chains, and which were named by him the chaplet cocci. These are now usually termed streptococci. But it was not till 1884 that Fehleisen determined accurately what he considered to be the specific streptococcus of this inflammation.

These two varieties of cocci (strepto- and staphylococci) are the usual causal agents in local inflammation and suppuration, and they were once regarded as specific pyogenetic (pus-forming) micro-organisms, but we now know that many other pathogenetic organisms can, under suitable conditions, give rise to local inflammation; and not to local inflammation only, but to suppuration also. I need only mention now the *Bacterium coli communis*, the *Bacillus typhosus*, the pneumococcus, and the *Bacillus pyocyaneus*. But probably *all* pathogenetic micro-organisms could, under suitable conditions, be led to cause a local inflammation, and all phlogogenetic organisms *can* give rise to suppuration.

Now in what way do the bacteria give rise to inflammation? There only exist two ways in which they can produce effects upon the tissues to which they gain an access. They can act either (1) mechanically as foreign particles, or (2) by means of chemical substances which they produce. Their action as foreign particles is exactly comparable to that of any other kind of foreign particulate matter which is insoluble, and therefore chemically inactive, and can only be purely mechanical in nature. The injury, if any, thus produced is purely mechanical injury, and as such will not call forth the inflammatory reaction, as we have already seen. If they gain access to the circulation they may give rise, like any other foreign particles, to blocking of small vessels, or embolism. For this to occur they must be present in enormous multitudes, and that is the limit of their possible effect excluding every form of chemical action. The inflammation they occasion must therefore be dependent on this latter action.

The chemical substances produced by micro-organisms are very various, and are by no means fully understood or even identified. I need only mention here two very important groups, the specific poisonous bodies, or so-called toxins, and the bacterial ferments. The former cause degeneration and destruction of living cells, while the latter exercise digestive action and lead to peptonisation of proteid material, and to a further breaking down with the production of bodies of the oxyphenyl series, which were at one time looked upon as antiseptics. This idea was largely wrong, but it had the great result of leading to the invaluable discovery of toxins and antitoxins, and the consequent initiation of modern serum-therapeutic measures. The peptonising action of bacterial ferments may probably afford an explanation of the fact that while the ordinary inflammatory exudations very readily coagulate, and usually undergo coagulation in the body, thus leading to a deposition of fibrin on the inflamed surfaces, the liquor puris of a suppurative inflammation will not coagulate, since its fibrinogen has been already peptonised within the abscess cavity by bacterial action.

The inflammatory reaction brought about by an invasion of bacteria is therefore due to the injurious action of the chemical substances which they produce. Accordingly their local action is

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exactly comparable to that of other chemical irritants, such as I have already mentioned, and to that of culture fluids from which the bacteria have been separated by filtering them through porcelain in the Chamberland filter.

The action of the chemical irritants, however, is in most cases chiefly, if not entirely, local, since as they gradually diffuse into surrounding parts they are diluted by the tissue fluids, and their activity is in consequence progressively diminished. But that of micro-organisms is not thus limited, since they are capable of independent growth, and if they reach the circulation may be carried to distant parts, where they become established and proliferate, and again produce specific harmful substances, thus giving rise to metastatic inflammations. Or without thus becoming localised in secondary foci of infection, they may survive and flourish in the blood, producing the condition known as septicæmia, which has received the appropriate name of "inflammation of the blood" from several of the French pathologists.

The chemical irritants are also able to produce a metastatic inflammation in parts where they collect in concentration, after diffusion through the body from the local focus of origin or point of introduction,—either by reason of a selective action and peculiar affinity for certain groups of cells, in which accordingly they gradually concentrate themselves; or because in the course of their destruction in the body, or in their excretion from it, they are collected into certain organs where accordingly they come to act in concentration. To give a few examples, the products of diphtheria bacilli diffusing through the body from the local lesion are excreted by the kidney, whose cells abstract them from the circulating blood. And if they reach these cells more rapidly than they can be discharged a local concentration will occur, which may result in the production of acute nephritis. Similarly certain products of diphtheria bacilli appear to have particular affinity for the nervous tissues, and may collect in these in quantity sufficient to produce neuritis with the familiar results. Tetanus toxin has a selective preference for nerve-cells, with which it enters into combination, and is thus accumulated in them as it passes to the circulation from the local focus of infection, until enough is present in the nervous centres to produce the picture of disease which we call tetanus. Cantharides, which is excreted by the kidney, may similarly produce albuminuria or actual nephritis when present in that organ in sufficient concentration; and mercury may have the same effect, especially in a kidney which has been already injured by disease. Alcohol may possibly exert a similar influence on the hepatic cells.

We may accordingly conclude, from the considerations I have put before you, that there is no *essential* difference between the inflammations which are brought about by micro-organisms and those produced by other agencies.

We now proceed to a consideration of the *rôle* played by the leucocytes in the inflammatory process.

We have already seen that the passage of the white corpuscles from the vessels of an inflamed part, and their accumulation in the inflammatory area, was an active process. What is the meaning of this phenomenon, and how is it produced? The leucocytes appear to be *attracted* from the neighbouring vessels and tissue spaces to the affected part. And since the inflammatory reaction is brought about by chemical agencies, the attraction of the leucocytes is presumably of chemical origin. It is accordingly spoken of as *chemotaxis*.

Phenomena of similar nature, which may be grouped with those of chemical attraction of the leucocytes under the general term *allurement*, were first observed by Engelmann, who found that if a fluid containing motile bacteria were illuminated in one part by beams of light of various colours, the rest of the fluid being exposed to diffused daylight, the bacteria were attracted by certain colours, as for example purple, so that they gradually collected in the part illuminated by that particular colour, and this phenomenon he termed *phototaxis*.

The events of chemotaxis were investigated by Stahl and Pfeiffer; and it was shown that certain substances exert attraction, while others are repellent for living protoplasm. These phases are usually now spoken of as positive and negative chemotaxis. Moreover it was found that living protoplasm can become habituated to previously repellent substances. Thus a plasmodium could be trained by gradually lessening its supply of water to send out processes into a salt solution which previously had exercised both a repellent and an injurious action on it.

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As regards leucocytes, it appeared from the experiments of Leber, Metchnikoff, Massart, Bordet, and others, that certain chemical solutions possess attractive power for these,—for instance, a solution of malic acid; while others, like quinine, exert repulsion. The same is true of certain slightly soluble metals, such as mercury and copper. Thus Leber showed that if a fine capillary tube containing mercury be carefully inserted in a vein it becomes rapidly filled with leucocytes, although the circulation has continued normal and no coagulation has occurred upon the tube. Further, a substance which in strong solution has repelled the leucocytes will, in sufficiently dilute solution, give the positive phase and cause attraction.

In the same way the soluble products of bacteria exert a chemotactic action more or less positive, according to the less or greater virulence of the micro-organism, the leucocytes approaching more readily the less virulent forms, and being but little attracted and perhaps even repelled by very virulent varieties. This is well shown in an experiment of Metchnikoff. If two cultures of anthrax bacilli be taken, the one virulent, the other attenuated, that is to say of diminished virulence, and one ear of a rabbit be inoculated from each culture, an intense inflammation is set up in both the ears. But while in the ear inoculated with attenuated anthrax there is very little serous exudation, but an immense accumulation of leucocytes, in the other there is a great effusion of clear serum containing hardly any leucocytes at all.

Dead tissue similarly acts as a positively chemotactic agent. Hence it follows that the leucocytic diapedesis of inflammation occurs as the result of the chemotactic action of the injured tissue, and of the chemical or bacterial irritant which has caused the injury. This affords an explanation of the fact that in purely mechanical injuries in which, comparatively speaking, very few cells are actually destroyed, and in aseptic destruction of cells in parts remote from vessels, as in the centre of the cornea, no diapedesis occurs—for here the products of cell disintegration never reach the circulation in sufficient concentration to produce a chemotactic influence, and no bacterial chemotactic substances are present.

Thus we see that while the inflammatory exudation is conditioned, at any rate chiefly, by the direct injury produced in the vessel walls by the action of the irritant or noxa, the determination of leucocytes to the area of inflammation is the effect of chemotactic action exercised by that irritant, or by the products of the cell destruction which it causes.

I have said that though a weak solution of a given substance may be positively chemotactic, yet a strong solution may be found to exercise a reverse effect. The same fact is probably true of the bacterial products. It would then follow that at the centre of inflammation, where they are present in concentration, they may be able to repel the leucocytes, though further off where they are weakened and diluted by their gradual diffusion they will have a positive chemotactic action. The bacterial products are in many cases also capable of destroying leucocytes which come within the area of their concentrated action.

These two factors suggest the explanation of the fact that in any inflammation due to a very virulent bacterium very few leucocytes are present in the inflammatory area, and even these are for the most part killed by the bacterial products, so that instead of the formation of a local abscess a spreading inflammation is produced.

Besides their action in producing chemical irritants the bacteria present themselves as foreign bodies, and as such they are subject to the same conditions as any other foreign particles.

Now throughout the animal series, from the unicellular protozoa upwards, it is a property of *amœboid cells* to take up particles suspended in the fluid in which they live by a process of ingestion. Around these particles, when ingested, there is developed, as can be seen in the amœba, a vacuole, and this contains a fluid which Le Dantéc showed is acid in reaction, and a peptonising ferment (Miss Greenwood, Krukenberg). The particles are thus digested, or if they are indigestible are subsequently extruded. To this process of ingestion Metchnikoff gave the name phagocytosis. The property of thus ingesting foreign particles is possessed in marked degree by certain varieties of the leucocytes, including the wandering cells of tissue spaces, and by certain fixed cells also, notably the endothelial cells which line the vessels and the serous cavities, to which the term *fixed* phagocytes is applied by Metchnikoff.

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The wandering phagocytes attracted to the inflammatory focus by a chemotactic influence approach the bacteria, and if they are not first destroyed by the bacterial products they rapidly ingest the micro-organisms themselves, and subsequently exert on them digestive action. In a precisely similar manner particles of other foreign matter and dead tissue undergo the process of ingestion and digestion by the phagocytes.

Cœlomic and non-phagocytic 1 and 3 Hæmal and phagocytic 2 and 4	1. Coarsely granular, with horse-shoe nucleus	Oxyphile. Stain with acid dyes	2 to 4 per cent. of leucocytes (eosinophile).
	2. Finely granular, with polymerous nucleus		20 to 70 per cent. of leucocytes.
	3. Coarsely granular, with round nucleus	Basophile. Stain with basic dyes	None in blood.
	4. Finely granular, with trilobed nucleus		1 to 5 per cent. of leucocytes, more numerous after meals than at other times.
Phagocytic	5. Hyaline, with kidney-shaped nucleus	Non-granular	2 per cent of leucocytes.
	6. Lymphocytes.		Up to 30 per cent. of leucocytes.

The fluid exudate itself may also have a destructive action on bacteria, as Nuttall showed to be the case with serum. This, as we shall see when we come to the discussion of infection, is probably brought about by ferments liberated by the destruction and disintegration of leucocytes.

Phagocytosis and the effect of chemotaxis are well shown in the following experiment of Leber in the frog. A fine suspension of vermilion is injected into the dorsal lymph sac of a frog, and after a short time some mercury is introduced into the anterior chamber of the eye. In a little while a bright vermilion colour can be seen in the cornea of this eye, produced by an accumulation of leucocytes which had taken up vermilion in the dorsal sac by phagocytic action, and have been attracted to the corneal spaces by the chemotactic influence of the mercury placed in the anterior chamber.

I have hitherto spoken of the leucocytes collectively without distinction of kind, as if all the varieties took an equal share in the phenomena of inflammation, but this is not the case. Not all the leucocytes are phagocytes; nor, on the other hand, is the power of phagocytic action limited to leucocytes, since, as we have seen, the cells of endothelia, among others, possess a similar activity.

Differences in kind among the leucocytes were first described by Wharton Jones in 1846. His observations were followed and extended by those of Rindfleisch, Max Schultze, and others. But a new era in the histology of blood was inaugurated by the discovery of Ehrlich of the differentiating value of the aniline dyes. He subdivided the leucocytes into five varieties, according to their *granulation* and their reaction to *acid* or *basic* stains respectively. His observations have been somewhat modified by the results of Sherrington, and of Kanthack and Hardy, who divided the wandering cells into the six following varieties:

Metchnikoff had previously, as the result of his researches on phagocytosis, subdivided the blood leucocytes into the following classes:

1. Lymphocytes.
2. Large hyaline mononuclear phagocytes—"macrophagocytes."
3. Smaller neutrophile polymorphonuclear phagocytes—"microphagocytes."
4. Eosinophile—coarsely granular oxyphile cells, non-phagocytic.

These two classifications correspond very closely in the main. We see from them that phagocytic leucocytes are those contained in Metchnikoff's Classes 2 and 3, and correspond respectively to Class 5 and Classes 2 and 4 combined of Kanthack's classification.

Besides these leucocytic phagocytes, certain other cells which belong developmentally to the same group of the mesodermal cells possess the phagocytic power. These are the "fixed cells" of connective-tissue spaces, and the endothelial cells lining the vessels and the serous cavities, the latter being simply greatly enlarged lymphatic spaces which communicate directly with the lymphatic vessels by means of so-called stomata. All these cells possess the faculty of forming pseudopodia, and of thus enclosing and ingesting foreign particulate material.

The information we have gained up to the present from our discussion of the phenomena of inflammation may now conveniently be re-

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capitulated by summarising briefly the events in the formation of acute abscesses. These have been carefully worked out by various observers, and especially by Hohnfeldt for staphylococcus (*Staphylococcus pyogenes aureus*) suppuration in the rabbit.

The vascular changes proceed exactly as in simple inflammation. Then within a few hours the leucocytes are found beginning to collect in the affected area. These are at first chiefly of the mononuclear variety, but after a short time leucocytes with polymerous nuclei become more numerous and predominate. At this stage it is seen that the massing of leucocytes, which has occurred at the focus of infection around the accumulation of proliferating cocci, is due to the collection in that region of enormous numbers of the multinuclear leucocytes, while in the boundary zone, where cocci are less numerous or absent, the uninuclear variety of leucocyte predominates.

After some forty-eight hours a well-defined local abscess has been formed, in which the following facts may be made out. The leucocytes within the abscess cavity are almost entirely of the polymorphonuclear variety; some of them are degenerating, and the normal tissues of the central area have broken down and been destroyed. There is no evidence of any process of repair, nor any multiplication by proliferation, either of the leucocytes or of connective-tissue cells. Most of the cocci are by this time intra-cellular,—that is, they have undergone phagocytosis. Towards the periphery of the abscess they are fewer and fewer in number, though some may be found free in the lymph spaces, and others are seen to lie within the endothelial cells of the blood-vessels, having been taken up by these by phagocytic action.

Destruction of leucocytes by bacterial action, fresh diapedesis from the vessels, and the phagocytic process go on continuously until at length the cocci are destroyed, but not until about the *tenth* day does any sign of *new formation* or regeneration of tissue appear. About this time the connective-tissue cells around the margin of the abscess may be seen to undergo proliferative changes and to multiply, giving rise to a formation of epithelioid cells which are called fibroblasts. Among these new capillaries are thrown out, and the abscess wall assumes the character of granulation tissue, from which cocci are altogether absent, and only very few leucocytes are to be seen within its spaces. Cicatrisation follows.

The changes which are found at different intervals after the inoculation of staphylococci may be more easily remembered from the following table:

At about four hours.—Local collection of mononuclear leucocytes; diapedesis of polymorphonuclear leucocytes; multiplication of the cocci.

After ten hours.—Polymorphonuclear leucocytes in the centre ingesting cocci; mononuclear leucocytes in the boundary zone; ingestion of cocci by endothelial and other fixed phagocytes.

After forty-eight hours.—Abscess packed with polymorphonuclear leucocytes and ingested cocci, destruction of the tissues of the central area. Some cocci seen outside the abscess either free or already ingested by the phagocytes.

From forty-eight hours onwards.—Degeneration of leucocytes; diapedesis; phagocytosis; destruction of cocci.

Tenth day onwards.—Proliferation of connective-tissue cells; formation of granulation tissue; cicatrisation.

Another termination of the infection is, however, possible. If the bacterium introduced is highly virulent it may produce a very rapid and wide-spread destruction of leucocytes in the affected area, or even perhaps hold them at a distance by exerting negative chemotaxis—though this is not entirely proved at present. Hence, as it multiplies, it is not held in check by phagocytic and digestive action, but can spread freely in the exudate, passing to neighbouring parts by contiguity of tissue, and being carried on in the lymph stream which leaves the area of inflammation. It thus becomes disseminated or sown throughout the body, and may give rise to general infection of a septicæmic or pyæmic character.

But this condition we must leave aside until we come to deal with the phenomena of infection.

WE have received from Messrs. J. and A. Churchill, of 7, Great Marlborough Street, a copy of the seventh edition of 'Materia Medica,' by Dr. Hale White, Lecturer on Medicine, and Physician to Guy's Hospital. We will publish presently a notice of this valuable work.

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